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Editorials

STATE MEDICINE A POLITICAL FOOTBALL

State Medicine invariably becomes involved in politics making the health of the nation a political football to be used by the particular party or clique which happens to be in control.

The whole theory of State Medicine or compulsory insurance is unsound economically. There has not yet been a scheme of State Medicine put into practice which gives good care to the indigent. They all apply only to those having employment. The fact is, that those who are employed could pay for their own medical care if they put their health before their pleasures and luxuries. The average family in America today pays more for tobacco alone, more for candy alone, more for liquor alone, than it pays to its family physician. The truth of this statement was very aptly expressed by Congressman Pettengill when he said, "We might as well look the fact squarely in the face that the drive for State Medicine finds its chief motive power in the desire to shift the economic burden involved from the shirker to the worker; the shiftless to the thrifty; the waster to the saver; the unfortunate to the fortunate; the drunkard to the sober; in short, to reap where others have sown."

Hubbard Prather Saunders, M. D.
(In Peoria Medical News).

CANCER IS CURABLE

The American College of Surgeons at the end of 1938 listed 29,195 people as cured of cancer. Cancer is costing the country $900,000,000 a year. Education of the public relative to the curability of cancer began in earnest only within the past few years. That a great deal of education still remains to be done was revealed recently by a survey of the American Institute of Public Opinion. This survey showed that more than one of every three Americans did not know that cancer is curable, if treated in time. Forty-one per cent thought cancer is contagious; and as to be expected, 75 per cent of the people

polled feared cancer more than any other disease. The public first began to learn of the curability of cancer when the American College of Surgeons conducted "Cancer Is Curable" symposium at their annual meeting in 1932. At the 1932 Congress thirty distinguished surgeons reported 8840 cancer cures with no recurrence of the disease five or more years after operation. Early diagnosis and treatment made the cures possible. Cancer is not likely to recur if there is no evidence of it after two years, and five years is set as an almost safety index.

The publicity resulting from the symposium conducted by the American College of Surgeons in 1932, removed inhibition about cancer and cancer cures. Shortly thereafter, lay education on symptoms and urgent need of prompt diagnosis and treatment received an impetus that has been carried through to the present time.

Through the public press, radio talks, lectures and other means of publicity intensive cancer campaigns have been carried on in every State. The campaign of publicity has in a large measure dispelled the fear that has hampered the efforts of doctors in the past. Twenty-nine thousand, one hundred and ninety five former victims of this disease is sufficient proof that cancer can be cured.

URGENT NEED FOR A UNITED PROFESSION

Medical conditions demand far seeing leadership. If medical integrity is to survive, its course must be steered off the rocky shoals of state and government dictatorship and competition. In union there is strength, if organized medicine will present a united front success is sure to crown the effort.

Not a home in the Nation that is not reached by some doctor during the course of the year; perhaps not an individual in the United States who is not met face to face and engaged in personal conversation by one of the medical or allied professions in a given twelve months.

What a power, if organized, would be the doctors and allied professions of the United States in combating medicinizing socialization schemes, regimentation of the profession, government dictation and practice of medicine, government in every kind of business and profession, scheme for health centers, clinics, health insurance and other tax supported medical service schemes.

Items that are of vital importance in helping to preserve future medical stability and to which we should direct our thoughts and best efforts are:

(1) That we continue in the future as we have in the past to fight for the economic welfare of the physician.

(2) That we lose no opportunity to advance the influence of organized medicine in civic affairs.

(3) That we build up the membership of organized medicine to the point of including all the ethical, legitimate doctors in the National organization.

(4) That we continue to oppose dictation, in any form, of the practice of medicine by lay organizations, as well as attempts by individuals, medical schools, institutions or corporations to supplant physicians in medical affairs or to compete with doctors in the practice of medicine, surgery and the specialties.

(5) That we promote efficiency in medical practice and the highest attainment possible in medical science.

(6) That we advocate unceasing activity on the part of the individual physician in order to secure the economic betterment and stability of organized medicine as well as individual members that make up the parent organization.

(7) That we stand four square for the conservation of the ideals and interests of doctors both individually and collectively.

(8) That we develop to the ninth degree contacts and relationships with civic bodies, to the end that we may be able to give them expert assistance and advice, and be able to explain to welfare units conclusions based on experience rather than on theory.

(9) That there should be an awakening to the fact that federal aid, lay interference and bureaucratic control are too big a price to pay in return for what we get.

(10) That we are whole-heartedly in favor of rigid enforcements of the code of ethics relative to publicity and propaganda, etc., by institutions, corporations, philanthropic or otherwise as is rigidly enforced upon individual physicians.

(11) That we emphatically maintain, that the legitimate function of organized society is limited to public health activities, the teaching of personal hygiene, rendering medical service to the inmates of charitable and penal institu-

tions and education of the public along the line of disease prevention.

(12) That we unreservedly deplore lay dictation and practice of medicine as being both an absurdity and an anomaly; that the care of the sick should be strictly under the supervision of scientific trained men; that the treatment of the sick is strictly a problem of the medical profession.

(13) That we are primarily opposed to any plan for giving medical service that brings into the picture of medical practice business methods and commercialism which are fatal to medical science and to any setup of medical service that will break down the relationship between doctor and patient or to any plan which will deteriorate medical service, inhibit medical advancements, and break down confidence in the medical profession.

(14) That we oppose any form of tax supported medicine which take increasingly from the individual more and more of the responsibility for his own existence and which enter increasingly into the affairs of human life.

(15) And finally, it must be universally understood, that there can be but one master in the house of medicine and that is the doctor himself.

MEDICAL WRITING, THE TECHNIQUE AND ART

Dr. Morris Fishbein in a useful and condensed handbook entitled "Medical Writing, the Technique and Art," recently off the press, tells why papers sent for publications are so frequently rejected. Want of space, subject overworked, too theoretical, are given as the most frequent reasons for rejection. If you have delivered an address before a medical society, it is apt to be prolix, wordy and hastily assembled—perhaps just a pot boiler gotten together for reading rather than printing. Such a paper is likely to meet the same fate. Dr. Fishbein thinks that "a manuscript that is fit to be read is sometimes fit to print, but that a manuscript fit to print is always fit to read." The editor does not like long papers and he devotes a whole chapter of his book to showing *how, by careful revision, most papers can be reduced by at least twenty-five per cent.*

"In his chapter on style the author enjoins simplicity and clearness, in his discussion of subject and material he gives admirable advice, particularly elaborating the preparation of the now much neglected case report. He discusses the detailed construction of the manuscript, title, words and phrases, spelling, capitalization, abbreviations, numbers, proper usage in the manner of pharmaceutical products and prescriptions, and how to secure a bibliography. With considerable detail he goes into the mechanical preparations of manuscripts, illustrations, charts, and tables. The author concludes with emphasis upon the importance of the revision of papers to be submitted for publication, *telling us that Sir William Osler always made at least three drafts of a manuscript, Sir Clifford Albutt four, and Anatole France eight, and leaving us with the impression that he rather agrees with Trelease and Yule who recommend ten revisions before submitting the result to the printer.*" (Italics Ours)

ILLINOIS PHYSICIANS PARTICIPATING IN THE A. M. A. SCIENTIFIC PROGRAM

The following Illinois physicians participated in the scientific program at the A. M. A. St. Louis convention, naming them as they appeared on the program:

Ernest E. Irons, moderator, discussion on Pneumonia; O. H. Robertson, etiology, experimental and general, pathology (infection; mechanism of invasion; bacterimia, variations, types, significance in relation to therapy).

Andrew C. Ivy, physiology of the Biliary Tract.

N. C. Gilbert, chairman, section on Practice of Medicine, and delivered the chairman's address for the section.

Emil Schleicher, a paper on "The Reaction of Periphere Blood and Bone Marrow in Chronic Hemorrhage and in Essential Thrombocytopenic Purpura" (lantern demonstration).

George K. Fenn, "The Mechanism of Death in Pulmonary Embolism" (lantern demonstration).

Clarence F. G. Brown, "An evaluation of Therapy of Peptic Ulcer."

M. Herbert Baker, "Treatment of Pneumonia" (lantern demonstration). Paul S. Rhoads discussed Dr. Baker's paper.

Loyal Davis & John Martin, "Surgical Lesions in the Paratrigeminal Area" (lantern demonstration).

Geza de Takats. Discussed a paper on "Throm-

bophlebitis: The Etiologic Factor of Vasospasm and the Treatment by Sympathetic Block" (lantern demonstration).

Lester R. Dragstedt. Discussed a paper on "The Immediate and Late Results of Surgical Treatment of Acute Perforations of Peptic Ulcer" (lantern demonstration).

Fred L. Adair and M. Edward Davis, presented a paper on "Atrophy of the Vulva" (lantern demonstration).

Charles E. Galloway. Discussed a paper on "The Responsibility of the Medical Profession in the Movement of Birth Control."

Franklin F. Snyder. Discussed a paper on "The Effects of Obstetric Analgesia on the New Born Infant" (lantern demonstration).

George H. Gardner. Discussed a paper on "Post Operative Care of the Urinary Bladder" (lantern demonstration).

Bernard Portis and Harold A. Roth. Presented a paper on "Diagnosis and Treatment of Hyperthyroidism."

F. H. Falls. Discussed the paper of Drs. Portis and Roth.

Edward D. Allen. Discussed a paper on "Urinary Incontinence and Relation to Cystocele" (lantern demonstration).

Sanford R. Gifford. Discussed a paper on "The Treatment of Secondary Glaucoma" (lantern demonstration).

Harry S. Gradle. Discussed a paper on "The Value of Early Operation in Chronic Primary Glaucoma" (lantern demonstration).

Percival Bailey. Discussed a paper on "Primary Tumors of the Optic Nerve: A Phenomenon of von Recklinghausen's Disease; a Clinical and Pathologic Study with a Report of Five Cases and a Review of the Literature" (lantern demonstration).

James E. Lebensohn. Discussed a paper on "Cataract Following Inhalation of Paradichlorobenzene" (lantern demonstration).

Edward V. L. Brown. Discussed a paper on "Ophthalmology's Place in the Prevention of Blindness."

Frank J. Novak, Jr. Discussed a paper on "Argyria Resulting from Intranasal Medication" (lantern demonstration).

Howard C. Ballenger. Discussed a paper on "Infection of the Mouth and Face" (lantern demonstration).

George E. Shambaugh, Jr. Presented a paper on "Diplacusis: A Localizing Symptom for Disease of the Organ of Corti; Theoretical Considerations, Clinical Observations and Practical Application" (lantern demonstration).

Heyworth N. Sanford. Presented a paper on "The Effect of Various Complementary Feedings on the Gain in Weight and Stimulation of Breast Milk During the Newborn Period" (lantern demonstration).

Willard O. Thompson and Norris J. Heckel. "Treatment with Male Sex Hormones." F. C. Koch, discussed the Thompson-Heckel paper.

Clarence F. G. Brown. Presented a paper on "An Evaluation of Therapy of Peptic Ulcer."

M. Herbert Baker. Presented a paper on "Treatment of Pneumonia" (lantern demonstration).

J. P. Simonds. Presented a paper on "Experimental Studies on the Pathogenesis of Nephritis" (lantern demonstration).

Richard B. Richter. Discussed a paper on "The Neurotropic Viruses" (lantern demonstration).

Arthur Weil. Discussed a paper on "Clinical Aspects of Vitamin B Deficiencies" (lantern demonstration).

George W. Hall. Discussed a paper on "The Post-Traumatic Psychoneurotic State and Traumatic Encephalopathy" (lantern demonstration).

David Slight. Discussed a paper on "The Differential Diagnosis of Anxiety Neurosis."

H. T. Carmichael and J. H. Massermann. Presented a paper on "Results of Treatment in a Psychiatric Outpatient Department" (lantern demonstration).

L. J. Pollock. Discussed paper on "Delayed Paralysis of Nerves from Indirect Trauma."

Kathleen B. Muir, Evangeline Stenhouse and S. William Becker. Presented a paper on "The Action of Sodium Thiosulfate, Thiophenylsulfonic Acid and Formaldehyde Sulfoxylate in Arsenical and Mercurial Poisoning in Rabbits."

Theodore Cornbleet. Presented a paper on "Sugar Metabolism of the Skin" (lantern demonstration).

James Herbert Mitchell. Discussed a paper on "Dermatitis of the Ear."

Francis E. Senear. Discussed a paper on "The Senear-Usher Syndrome."

Charles M. McKenna. Presented a paper on "Hypospadias (in the Male): The Operative Technic for the Correction of Penile Deformity

and Reconstruction of the Urethra" (lantern demonstration).

Herman L. Kretchmer and Ralph C. Brown. Presented a paper on "Do Alkalis Used in the Treatment of Peptic Ulcer Cause Kidney Stones" (lantern demonstration).

E. J. Berkheiser. Presented a paper on "Excision of Patella in Cases of Hypertrophic Arthritis" (lantern demonstration).

Fremont A. Chandler and John R. Norcross. Presented a paper on "Sympathicoblatoma; A Report of Four Cases."

Edward L. Compere, Samuel W. Banks and William N. Krigsten. Presented a paper on "Regeneration of the Growth Epiphyses of Long Bones" (lantern demonstration).

Philip Lewin. Discussed a paper on "Low Back Pain: Correlation of the Signs and Symptoms of the Various Noninfectious Lesions" (lantern demonstration).

Edwin W. Ryerson. Presented a paper on "Excision of Scapula; Report of a Case Showing Excellent Functional Result" (lantern demonstration).

Walter L. Palmer. Discussion of a paper on "The Use of Hydrated Trisilicate of Magnesium for Peptic Ulcer."

Edward L. Jenkinson and Arthur F. Hunter. Presented a paper on "Bronchiogenic Carcinoma: A Diagnostic Enigma" (lantern demonstration).

Charles J. Betlach. Discussion of a paper "The Effect of Certain Anesthetics on the Blood."

OBSTETRICAL DEFINITION

(Formulated by The American Public Health Association and Endorsed by The Committee on Neonatal Morbidity and Mortality of The American Pediatric Society.)

1. Complete birth. A birth is complete the very instant of complete separation of the body of the infant from the body of its mother (regardless of whether or not the cord is cut, or the placenta detached).

2. Live birth. An infant exhibiting life after a "complete birth." The three evidences of life are: (a) breathing, (b) heart action or (c) movements of a voluntary muscle.

3. Still birth. An infant which does not exhibit evidence of life after a "complete birth": (no breathing, no heart action or no movement of a voluntary muscle).

4. Abortion. Any product of conception less than 28 weeks' duration, measuring 35 cm. or less, and weighing less than 1,500 grams (3¼ lb.).

5. Premature infant. An infant with a birth weight of 2,500 grams (5½ lb.) or less, with a "crown-heel" length of 47 cm. or less and a gestation of 37 weeks or less: the birth weight being the most important factor.

6. Neonatal period. The first 30 days of the infant's life. It is during these first 30 days that the mortality of all infant deaths under one year is the greatest (67 per cent). The usual causes, usually preventable, are prematurity, birth injuries and sepsis.

ANTEPARTUM CARE: CLINICAL LECTURE AT SAN FRANCISCO SESSION

Otto H. Schwarz, St. Louis (Journal A. M. A., Oct. 15, 1938), emphasizes the more important points that antepartum care involves. It is obvious that the patient must report as early as possible in pregnancy. A thorough general physical examination should be carried out early in pregnancy in order to recognize or eliminate medical complications. The medical complications that give most concern are heart disease, tuberculosis, diabetes and hyperthyroidism. They are in themselves problems, but superimposed on pregnancy they represent a serious picture and each case becomes an individual problem which must be carefully managed. Repeated pregnancies with medical complications should definitely be avoided. Although the mother may go through one pregnancy with little additional risk, this does not usually hold for repeated pregnancies. It is important to recognize pelvic abnormalities early in pregnancy; not only the contracted pelvis but the presence of pelvic tumors should be determined because, if intervention is indicated, they can best be handled early in pregnancy. The prevention of serious cases of toxemia of pregnancy and eclampsia can be readily accomplished by the proper antepartum care.

THE CHEMISTRY OF VITAMIN C

C. G. King, Pittsburgh (Journal A. M. A., Oct. 15, 1938), states that a primary requisite for studying the chemical nature of vitamin C was provided by Holst and Frölich when they observed that the guinea pig could be used as an experimental animal for the study of scurvy. Attempts to isolate the vitamin from natural products encountered great difficulty because of its extreme sensitiveness to destruction by oxidation. Isolation of vitamin C resulted, in one laboratory, from four years of systematic fractionation of lemon juice, based entirely on biologic assays, and that in another laboratory biologic assay for vitamin function was reported five years subsequent to the isolation of the vitamin as hexuronic acid, a silver nitrate-reducing agent in tissues, and two years after its negative assay as a hormone. The structural formula of the vitamin, type reactions and physicochemical properties are considered.

A two-year-old infant grew philosophical. "If I had my life to live over again," he remarked, "I would be a bottle baby so that there wouldn't be ashes falling into my eyes all the time."—*Med. World.*

MEDICAL ECONOMICS

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The reaction of the national press to the decision of Judge Proctor of The Federal District Court in Washington has been most favorable to the medical profession. The J. A. M. A. of August 12 presented editorials from different parts of the Nation and practically all of them agreed that the decision declaring the practice of medicine a profession and not a trade, was as expected and thereby the issue had been definitely clarified. Even the threat of reference to the Supreme Court has in no way changed the general impression that the medical profession has been exonerated of all suspicion of violation of the Sherman Act. One of the best articles on the decision was presented by *The United States News* of July 31 under the heading "Medicine Not a Trade—Anti-Trust Suit Checked." The first sentence read "The first important stop-sign in the Government's anti-trust crusade was a twelve page Federal Court decision." This was followed by a thorough review of the story of the suit and the reading of the article is well worth the time of any medical man interested in the details of the story. In other articles and editorials the opinion is quite freely expressed that the decision will have a salutatory effect on the other activities of the Anti-trust division of the Department of Justice.

If the decision has stopped further premature attempts to coerce the professions into friendly agreements entered into to avoid prosecution, or possibly persecutions as some of the articles intimated, the fight of the medical profession may eventually be one of the really important ones of this decade.

As intimated in the last article in this column, the plan of the Farm Security Administration in North Dakota for furnishing medical care to their clients in North Dakota has had considerable trouble and the opinion expressed by many of the thinking men of the profession that the money allotted was entirely inadequate has been verified. In order to save their face with the higher ups, as well as their clients and possibly the medical profession of at least North Dakota, the annual allotment has been increased considerably and an attempt is now being made to reenlist the medical profession of the state in the new plan. That this is not meeting with the approval of all the medical profession of the state is proved by a letter published in *The Victor News* for August, 1939, on page 5. This letter will follow in this column, immediately after this article. The contents will definitely answer the question in the minds of many of us as to the success of the plan in North Dakota. On page 49 of *Modern Medicine* for August appears an article entitled "Local Treatment Versus National Panaceas," which tells the results of experiments in North Dakota, Wisconsin, California and Australia in furnishing the low income group with medical care either under governmental or medical society supervision. The article is most critical of the result obtained under governmental control. If this article is still available, take time to read it.

Another article containing food for thought as to results of Group Hospitalization plan is to be found on page 28 of the August issue of *Medical Economics* on the subject "The Crisis in Group Hospitalization." With the general acceptance of the plan by both the laity and the medical profession as well as the phenomenal growth of the associations, it is well to consider some of the apparent difficulties of the plan and its successful administration. It has been the common opinion of both its opponents and proponents that an epidemic similar to that of Influenza in 1918 would during the first few years of any such plan result in financial crises, more severe the sooner they arrived and the less the surplus funds available at that time. Few had considered the dangers discussed in this article, although from the start the majority of the medical profession had been opposed to the including of any form of medical care in the hospitalization plan. But apparently it has been

included in some of the plans with results exactly as feared by the medical profession.

Current medical journals continue to report various plans for furnishing medical care on a pre-payment plan in various parts of the nation. It is too early to report definitely on the success or failure of any plan. Many states are trying out several different plans in different cities within their boundaries and in the next few years they should be able to outline definite successful plans. In accord with the instructions received at the meeting of the House of Delegates in Rockford in May, 1939, this problem is receiving much study by several committees of the Illinois State Medical Society. Their conclusions will be correlated by a special Committee from the Medical Economics Committee who expect to have a definite report ready at the next meeting of the House. Meanwhile, plans in nearby states are being investigated either by correspondence or by visiting the same, so that the success or failure can be checked from all angles. Much of the time of the last meeting of the Council was given over to consideration of this problem. All of the time of the meeting of this Committee, which was held immediately following adjournment of the Council was given to this problem. We must remember that the American Medical Association report that there are over a thousand such plans in operation in the United States at this time. It is of course impossible to study all or even most of them, but enough can be studied to select the successful portions of the best plans to use the same in any plan to be outlined for consideration at the next annual meeting. If those of you, who have plans or ideas on the subject, will get in touch with some member of this committee, whose names will be found at the top of this column and talk over your ideas with said member, I am sure that it will be to the mutual advantage of all concerned.

Meanwhile, remember that your Senators and Representatives are at home looking over their political fences. Take time off and let them know that you are one of their constituents and that you have definite ideas as to what you want done down at Washington, particularly as it affects the medical profession. They will be glad to listen to you. That is one of the reasons they are home. But go to them prepared with definite facts and reasons why Senate Bill 1620 should be defeated.

E. S. Hamilton, M. D.,
Chairman.

NORTH DAKOTA DOCTOR SPEAKS

To the Editor:

In your July number of VICTOR NEWS, under *News Flashes,* I notice an article with the heading of Washington, referring to the F. S. A. health program in North Dakota, and the concluding sentence, "North Dakota Medical men are reported eager to have the new plan start."

The propaganda put out by Washington nowadays from these various agencies under the federal government's supervision is each day resembling more and more the propaganda spread about by the dictator countries of Europe.

I venture to say that there is not one in ten M. D.'s in the state of North Dakota who is eager to have anything like the last F. S. A. setup start again in this state. We were only too glad to see that one come to an end, and are hoping that we shall never see the like again.

I just wanted to let you know how most of the doctors in North Dakota feel so that you will not be misled by articles such as that coming out of Washington. If they really want an opinion so that it can be published they might ask a few doctors of the state before drawing any hasty conclusions.

(Signed) R. D. Nierling, M. D.,
Jamestown, North Dakota.

Editor's Note: VICTOR NEWS is always glad to publish the opinions, such as Dr. Nierling's, of members of the medical profession concerning articles furnished to or prepared by us for publication.—Victor News, August, 1939.

Correspondence

THE COLUMNISTS COMMENT ON THE ARNOLD PRONUNCIAMENTO

Paul Mallon, widely circulated by King Features Syndicate, in his column News Behind the News, after describing the case and stating that if the American Medical Association is a monopoly so is the American Bar Association, says that this suit—

would simply mean the Justice Department is misnamed and has become instead a prosecution department to enforce, not alone the law, but social and economic theories.

Mr. Mallon also indicated that application of this principle would mean that newspaper correspondents might object because some of them cannot get into the press galleries of Congress, and the attendance on the President's press conferences is also strictly limited. Those who can get in have a great commercial advantage over those who cannot.

David Lawrence, widely syndicated, feels that—

the doctors have a society which is as much entitled to protection under the Wagner Relations Act as any other association.

After explaining his view that the Department of Justice has intervened principally to help the Group Health Association, Inc., Mr. Lawrence says,

The attempt to drag the antitrust laws into a controversy over what is or is not proper medical care is a piece of amazing political stupidity, but it is another example of how the zealots in the administration, with the full approval of President Roosevelt, are manipulating the laws of the United States to gain the goals of their so-called social experiments.

Mrs. Walter Ferguson, after explaining the nature of the controversy in Washington, says:

The average layman holds himself aloof from the controversy about his welfare now raging between the federal government and the American Medical Association.

If he is a middle-class citizen, he considers himself a martyr and is fond of saying that only the very rich and the very poor receive any consideration from doctors—although I doubt whether he would be willing to exchange places with the poor.

If we are honest, we must admit that our attitude toward the doctor has always been irritable and without consistency. We're ready to spend money on everything except our health. Most families will make any sacrifice to buy an automobile on the instalment plan, while they feel much aggrieved if they have to invest monthly sums to pay for medical attention which may have kept one of them out of the grave.

In the Baltimore Sun Mr. Henry L. Mencken says:

A defect common to all the plans so far proposed is that they are confined to employed persons and offer no aid to the unemployed. The latter are thus thrown on the free clinics, which are mainly manned by doctors who get nothing for their work.

This puts a very heavy burden on the medical profession, and there are doctors who begin to find it almost intolerable. Proposals have been made that the federal government offer them some remuneration, and no doubt this will be done as soon as the New Deal wizards can get around to it.

But whether the doctors will accept remains to be seen. Most of them are unalterably opposed to going on the public pay roll and so submitting their work and their fortunes to the will and whim of politicians.

Their opposition is hardly likely to be lessened by the fact that all the Communist organs have begun a violent campaign against them, denouncing them as sharks and scoundrels. Or by the fact that the New Deal has now joined in.

Bugs Baer, in a few succinct sentences, puts his finger squarely on the political motives involved in the attempt to make medical care the issue in future political campaigns:

The doctors don't approve of the federal health plan subject to a prescription by thirty-six states. A Democratic pharmacist deciphering a Republican doctor's diagnosis might mix in too much filibuster and not enough relief.

Health shouldn't be subject to politics. And when we're sick we don't want the opposition voting on it.

The insurance companies are nonpartisan. They pay off like a slot machine either way.

Journal A. M. A. August 13, 1939, says:

Elsewhere in this issue appear editorials published in various newspapers relative to the pronunciamento of Thurman Arnold, who proposes to determine whether or not the American Medical Association is a monopoly. Seldom has an action taken by the Department of Justice aroused the public interest and attention developed by this action. In addition to the editorials which have appeared in the newspapers, practically every one of the columnists syndicated in newspapers throughout the country has had something to say on the subject.

APPROVED LABORATORIES

To the Editor:

Enclosed is a copy of the text of (1) the new law requiring physicians to take blood specimens for tests for syphilis from all pregnant women under their professional care and (2) the amended law requiring pre-nuptial physicial examinations. Also enclosed is a list of approved diagnostic laboratories.

May I suggest that you publish the text of both laws and the list of approved laboratories in an early issue of the JOURNAL.

First, all laboratory tests required by these laws, in order to be valid, must be made in laboratories *approved* for that purpose by the State Department of Public Health and the kind of test must be one approved by that Department. *Second,* all cases of infection detected must be reported to the Department. *Third,* a marriage license may be issued to a person with evidence of infection which is considered non-communicable by the physician only with the approval of the State Director of Public Health who is required to investigate the situation before giving such approval. Exceptions to this are (1) pregnant women and (2) the mothers of living illegitimate children whose fathers are parties to the proposed marriages.

Physicians are required, moreover, to state in writing on each certificate of birth or stillbirth whether or not a blood test for syphilis was done on the mother of the child. This statement may be made on the margin of certificate forms now on hand. Future printings will have a special place and form for this statement.

I believe that to publish the texts of the two laws and to emphasize especially the points referred to above will be of considerable value to the medical profession of the State.

Thanking you for your consideration of this matter, I am

Very truly yours,
A. C. Baxter, M.D.,
Director of Public Health.

STATE OF ILLINOIS
DEPARTMENT OF PUBLIC HEALTH

August 5, 1939.

NEW LAWS FOR COMBATTING SYPHILIS

A law requiring professional attendants to take blood specimens for laboratory tests for syphilis from pregnant women and an amendment to the law which requires prenuptial physical examinations were enacted by the General Assembly which adjourned on June 30, 1939. Both of these laws are now in full force and effect. The text of each is reproduced below.

TEXT OF LAW ON PRENATAL BLOOD TESTS

Section 1. Every physician, or other person, attending in a professional capacity a pregnant woman in Illinois, shall take or cause to be taken a sample of blood of such woman at the time of the first examination. Said blood specimen shall be submitted to a laboratory approved by the State Department of Public Health for a serological test for syphilis approved by the State Department of Public Health. In the event that any such test shall show a positive or doubtful result a second test shall be made. Such serological test or tests shall, upon request of any physician in the State, be made free of charge by the State Department of Public Health or the Health Departments of cities, villages and incorporated towns maintaining Health Departments.

Sec. 2. In reporting every birth or still birth, physicians and others required to make such reports shall state on the birth certificate or still birth certificate, as the case may be, whether a blood test for syphilis has been made upon a specimen of blood taken from the woman who bore the child for which a birth or still birth certificate is filed, together with the date when the blood specimen was taken and the name of the laboratory making the test. In no event shall the birth or still birth certificate state the result of the test.

Sec. 3. This act shall be administered by the State Department of Public Health.

TEXT OF AMENDED LAW ON PRENUPTIAL EXAMINATIONS

Section 1. Section 6a of "An Act to revise the law in relation to marriages," approved February 27, 1874, as amended, is amended to read as follows:

Sec. 6a. All persons making application for a license to marry shall at any time within fifteen (15) days prior to such application be examined by a physician duly licensed in this State as to the existence of or freedom from any venereal disease, and, except as otherwise herein provided, it shall be unlawful for the county clerk of any county to issue a license to marry to any person who fails to present for filing with such county clerk a certificate signed by such physician setting forth that such person to the proposed marriage is free from venereal diseases as nearly as can be determined by a thorough physical examination and such standard microscopic and serological tests as are necessary for the discovery of venereal diseases. If, on the basis of negative laboratory and clinical findings the physician in attendance finds no evidence of venereal diseases, he shall issue a certificate to that effect to the examinee, which certificate shall read as follows, to-wit:

I, (Name of Physician).....being a physician, legally licensed to practice in the State of..... (my credentials being filed in the office of..... in the City of .....County of .....State of .....) do certify that I did on the ..... day of ....., 19...., make a thorough examination of ..... and considered the result of a microscopical examination for gonococci

and an approved serological test for syphilis, which was made at my request, and believe..... to be free from all venereal diseases.

....., *Signature of Physician.*

Such certificate of negative findings as to each of the parties to a proposed marriage to which laboratory reports of microscopical examinations of smears from the genitalia for the gonococcus of gonorrhea and serologic tests for syphilis are attached, shall be filed with the county clerk of the county wherein the marriage is to be solemnized at the time application is made for a license to marry. Laboratory tests for venereal diseases required hereunder shall be tests approved by the State Department of Public Health and shall be made by laboratories of said Department or by such other laboratories as are approved by said Department. Such tests as may be made by the health departments of cities, villages and incorporated towns maintaining laboratories shall be free of charge. The results of all laboratory tests shall be reported on standard forms prescribed by the State Department of Public Health.

Irrespective of the results of laboratory tests and clinical examination, the clerks of the respective counties shall issue a marriage license to parties to a proposed marriage (a) when the woman is pregnant at the time of such application, and (b) when the woman has, prior to the time of application, given birth to an illegitimate child which is living at the time of such application and the man making such application makes affidavit that he is the father of such illegitimate child. The county clerk shall, in lieu of the health certificate required hereunder, accept, as the case may be, either an affidavit on a form prescribed by the State Department of Public Health, signed by a physician duly licensed in this State, stating that the woman is pregnant, or a copy of the birth record of the illegitimate child, if one is available in this State, or if such birth record is not available, an affidavit signed by the woman that she is the mother of such child.

Also irrespective of the results of laboratory tests and clinical examination, the clerks of the respective counties shall issue a marriage license to parties to a proposed marriage when, after investigation, the Director of the State Department of Public Health, or his duly authorized representative, issues or causes to be issued a certificate that such marriage may be consummated without serious danger to the health of either party to the proposed marriage or to any issue of such marriage.

Any county clerk who shall unlawfully issue a license to marry to any person who fails to present for filing the certificate provided for in this Act or who shall refuse to issue a license to marry to any person legally entitled thereto under this Act, or any physician who shall knowingly and wilfully make any false statement in the certificate, or any party or parties having knowledge of any matter relating or pertaining to the examination of any applicant for license to marry, who shall disclose the same, or any portion thereof, except as may be required by law, shall upon proof thereof be punished by a fine of not less than $100.00 nor more than $500.00 for each and every offense.

Any person who shall obtain any license to marry contrary to the provisions of this section shall, upon conviction thereof, be punished by a fine of not less than $100.00 or by imprisonment in the county jail for not less than three (3) months or by both such fine and imprisonment.

Any license to marry issued hereunder, shall be void thirty (30) days after the date thereof.

APPROVED DIAGNOSTIC LABORATORIES

The diagnostic laboratories at the institutions listed below have been approved by the State Department of Public Health of Illinois for making the Kahn blood test for syphilis and microscopic examinations of smears for gonococci:

entitled thereto under this Act, or any physician who

Alton—Memorial Hospital, Rock Springs Drive; St. Joseph's Hospital, 915 E. Fifth St.

Belleville—St. Elizabeth's Hospital, 328 West Lincoln.

Cairo—St. Mary's Hospital, 2025 Walnut Street.

Canton—The Coleman Clinic, 24 N. Main Street.

Danville—Lake View Hospital, 812 N. Logan Street.

Decatur—Decatur and Macon County Hospital, End of N. Edward Street; St. Mary's Hospital, 220 South Webster Street.

E. St. Louis—East Side Health Dist., 325 East Broadway; St. Mary's Hospital Clin. Lab., 129 North 8th Street.

Elmhurst—Elmhurst Com. Hospital, 189 Avon Road.

Evanston—Church St. Clin. Lab., 636 Church Street; City Health Department, 1806 Maple Avenue; Evanston Hospital, 2650 Ridge Avenue.

Freeport—Deaconess Hospital, 420 S. Harlem Avenue.

Highland Pk.—Highland Pk. Hospital, 645 Homewood Avenue.

Jacksonville—Our Savior's Hospital, 446 East State Street.

Joliet—Prescription Shop Lab., 317 Morris Building.

Joliet—St. Joseph's Hospital, 372 N. Broadway.

Melrose Pk.—Westlake Hospital, 612 N. 12th Avenue.

Moline—Lutheran Hospital, 5th Avenue and 5th Street.

Oak Park—L. R. Hill Clin. Lab., 1011 Lake Street; Oak Park Hospital, 525 Wisconsin Avenue; West Suburban Hospital, 518 N. Austin Blvd.

Ottawa—Ottawa Clinical Lab., 112 Madison Street; Ryburn Memorial Hospital, 701 Clinton Street.

Peoria—Caterpillar Tr. Co. Lab., East Washington Street; Collins Clinic Lab., 427 Jefferson Building; Methodist Hospital, 221 N. Glen Oak Avenue.

Quincy—Quincy Clinic Lab., 1416 Maine Street.

Robinson—Brooks & Teasley Clin., New Otley Building.

Rockford—Rockford Health Dept., Walnut and First Streets; Rockford Hospital, Court and Chestnut Streets.

Springfield—St. John's Hospital, 7th and Mason Streets.

Urbana—Mercy Hospital Clin. Lab., Wright and Park Streets.

Waukegan—Victory Memorial Hospital, 1324 Sheridan Road.

CHICAGO

Acme Clinical Laboratory, 7910 S. Cottage Grove Avenue.

American Hospital Laboratory, 850 West Irving Park Blvd.

Augustana Hospital Pathology Lab., 411 Dickens Avenue.

Belmont Hospital Laboratory, 4058 Melrose Avenue.

Bethany Sanitarium and Hospital Lab., 3420 West Van Buren Street.

Burlington Medical Dept. Lab., 547 Jackson Blvd.

Central X-ray and Clinical Lab., 58 East Washington Street.

Chicago Board of Health Lab., 54 West Hubbard Street.

Chicago Laboratory, 25 East Washington Street.

Chicago Medical School Lab., 710 South Wolcott Avenue.

Children's Memorial Hospital Lab., 735 Fullerton Avenue.

Civic Medical Center of Chicago, 20 East Jackson Boulevard.

Clinical Laboratory, 1180 East 63rd Street.

Clinical and x-ray Laboratories, 818 East 47th Street.

Cook County Hospital Lab., 1828 West Polk Street.

Samuel Deutsch Serum Center Lab., 2912 Ellis Avenue.

Devon Laboratory, 6355 Broadway.

Edgewater Hospital Laboratory, 5700 North Ashland Avenue.

Englewood Hospital Laboratory, 6001 South Green Street.

Evangelical Hospital Laboratory, 5421 South Morgan Street.

Franklin Boulevard Hospital Lab., 3230 West Franklin Blvd.

Garfield Park Community Hospital Lab., 3821 West Washington Blvd.

Hyde Park Clinical Laboratory, 1525 East 53rd Street.

Illinois Central Hospital Lab., 5800 South Stony Island Ave.

Illinois Clinical Laboratory, 185 North Wabash Avenue.

Lawndale Laboratory, 3653 West 26th Street.

Lincoln Clinical & X-ray Lab., 2373 North Lincoln Avenue.

Lincoln Gardner Laboratory, 30 North Michigan Avenue.

Lutheran Deaconess Hospital Lab., 1138 North Leavitt Avenue.

Madison-Kedzie Clinical Lab., 9 South Kedzie Avenue.

Moore Clinical Laboratory, 55 East Washington Street.

Mt. Sinai Hospital Pathological Lab., 2750 West 15th Place.

John B. Murphy Hospital Lab., 620 West Belmont Avenue.

Northwestern Medical School Lab., 303 East Chicago Avenue.

Norwegian-American Hospital Lab., 1044 North Francisco Avenue.

Passavant Hospital Clinical Lab., 303 East Superior Street.

Quade Clinical and X-ray Lab., 6255 South Ashland Avenue.

Research & Educational Hosp. Lab., 808 South Wood Street.

St. Anne's Hospital Clinical Lab., 4950 Thomas Street.

St. Anthony dePadua Hospital Lab., 19th Street and Marshall Blvd.

St. Bernard's Hospital Laboratory, 6337 Harvard Avenue.

St. Elizabeth's Hospital Lab., 1431 North Claremont Avenue.

St. Luke's Hospital Laboratory, 1439 South Michigan Avenue.

St. Mary of Nazareth Hospital Lab., 1120 North Leavitt Avenue.

Scientific Pathological Lab., 6 North Michigan Avenue.

Walther Memorial Hospital Lab., 1116 North Kedzie Avenue.

Wesley Memorial Hospital Lab., 2449 South Dearborn Street.

Woodlawn Clinic Laboratory, 826 East 61st Street.

Woodlawn Hospital Laboratory, 6060 South Drexel Avenue.

New lists of approved laboratories will be published from time to time as others apply and qualify.

There are known to be 141 other local diagnostic laboratories which have not been approved. Of these 101 offer to do blood tests for syphilis and all, presumably, offer to do microscopic tests for gonorrhea.

The diagnostic laboratories of the State Department of Public Health have been approved by the National Institute of Health for doing all of the diagnostic tests offered.

CRITICISM OF THE WAGNER HOSPITAL BILL

The Journal of the American Medical Association offers the following criticism:

"Under section 1203 (a) (1), financial participation by the states is required. Naturally the extent of this participation will vary from state to state. Assuming, however, that the contributions of the Federal Government will be on a fifty-fifty basis, there will be available for the construction and improvement of government-owned general hospitals $16,000,000 in the fiscal year ending June 30, 1940, $100,000,000 in 1941 and $200,000,000 in 1942. Taking $4,000 as the average cost per bed of general hospitals, this bill would make provision for the addition of 4,000 general hospital beds in 1940,

25,000 general hospital beds in 1941 and 50,000 general hospital beds in 1942. These figures relate only to government owned hospitals and do not include such enterprises, public or private, as may be undertaken without the stimulus of a Federal subsidy.

"Over the eleven-year period 1928-1938 inclusive the average annual rate of increase in the number of beds in general hospitals was 1.9 per cent. The increases proposed in the Wagner Bill amount to a total of 79,000 beds, 16.2 per cent, in three years, or an average rate of increase of 5.4 per cent. In 1938 the general hospitals of the country were filled to 68.9 per cent of their capacity; 31.1 per cent of the beds were unused. Wherein lies the justification of the proposal to multiply threefold the normal increase of hospital facilities?"

"Section 1201 of the Wagner Bill, s. 1620 authorizes the appropriation in successive years of eight, fifty and one hundred million dollars, respectively, for the construction and improvement of general hospitals."

SOCIALIZED MEDICINE FROM A LAYMAN'S VIEW POINT

It would seem that Prof. Albert Gaylord Hart has overlooked some very important facts and some very serious objections to socialized medicine. Material things can be socialized, for good or for worse (mostly the latter), but brains and mental ability cannot.

Socialized medicine, for example, would only get the mediocre ability, or worse. No smart young man is going to spend the best years of his life studying medicine if he is denied his dream of fame and fortune.

The attempted prosecution of the American Medical Association was an ill-starred adventure and a criminal waste of taxpayers' money. If the same amount of money had been spent intelligently for medical service in state and county hospitals, or in municipal hospitals, the taxpayers' money would not have been spent in vain. Any bright young lawyer just out of law school knows why any professional association is not a trust or a monopoly under the statutes or within the meaning of the law.

It may be time to keep government doctors out of the American Medical Association, just as some bar associations are considering the denial of membership to government lawyers, for such organizations should be based upon private practice, exclusively, with no taint of politics.

A. F. Durand,
(Not an M. D.)
—Chicago Daily News.

NOW SEEK TO SOCIALIZE LAW, SAYS BOAKE CARTER

Are the lawyers to be next on the list to feel the heavy hand of government competition?

Solicitor-General Bob Jackson claims that lawyers in general are charging too high fees for their services and that the Government may therefore be compelled to provide free legal service for people who cannot pay the fees of the private law firms.

"Low cost, high volume" legal service was his solution.

This piece of advice, coming from a lawyer representing a political organization whose high jinks have been so confusing as to provide more work for the legal fraternity than at any other time in the history of the American bar, borders on the comic.

A lawyer is justified in charging a fee which he believes commensurate with the worth of the services he renders, as is any artist, radio singer, movie actor, engineer, doctor, dentist or other professional man.

We have not yet reached the stage where we must submit to a system of socialized legal advice, as Mr. Jackson seems to envision as America's crying need, any more than we are prepared to accept socialized medicine. The relationship between client and lawyer is as intimate as the relationship between doctor and patient. And the very first person to turn thumbs down on the idea of government competition with the lawyers would be the average American citizen himself.

To be sure, some of the lawyers charge fees which seem outlandish. But their "outlandishness" can be measured only by the worth of the service rendered by the lawyer in the estimation of the client. A man accused of murder may feel he paid a cheap price in $25,000 if the lawyer to whom he paid it convinced the jury his client was innocent. There are lawyers who specialize in certain departments of law.

Many of these establish a high standard of success before the bar. They are successful primarily because they are diligent and careful of detail. These characteristics are attainable only through long hours of hard work. For such "crimes" are they to be penalized?

Mr. Jackson was not known as anything extra-superspecial in the legal profession before he managed to climb aboard the New Deal bandwagon. By his avidity for politics and knowing whose political mug to kss, rather than by his technological cunning as a lawyer, did Mr. Jackson rise to the position of Solicitor-General. Not in the widest stretch of imagination could Mr. Jackson hold a professional candle to the standards of sheer legal brilliance of the late Solicitor-General James M. Beck.

Where, then, does Mr. Jackson obtain the presumption blandly to inform some of the ablest legal brains of the Nation that they had better socialize their talents or else risk government competition in free advice? Simply that he is a politically appointed Solicitor-General gives him no excuse.

Furthermore, the boom in the law business during the last seven years is traceable to one source and one source alone—the New Deal, which Mr. Jackson represents. Most lawyers nowadays have to be mental trapeze artists to keep abreast of the twisted concoctions that have poured forth in a steady stream from the legal brains employed by the New Deal.

"The Government," says Mr. Jackson, "sees a large number of citizens who help pay taxes, deprived of legal service because they cannot pay the provisional scale of prices."

To begin with, a citizen cannot turn around nowadays, figuratively speaking, without being forced to go to a lawyer to find out whether he is or isn't unwittingly breaking some fool law, government ruling or regulation. In the second place, the lawyer whom he consults has a perpetual nightmare himself trying to keep up with the parade. The average citizen would not always be forced to run for legal advice these days, as indeed he must to keep out of a government jail, if the master minds in Washington quit thinking up new methods of regulating American economy from soup to nuts.

"It has been for centuries thought the duty of government to take affirmative steps to see that its citizens received justice," Jackson observed.

The first step to carry out that thought in a "representative" form of government (Jackson forgot to include that word "representative") is for the Government to obstain from competing with the endeavors of its citizens!

Mr. Jackson had better go back to Jamestown, N. Y., and private law. He might get back to earth again—on the other side of the fence!—San Francisco *Examiner*, July 17.

SCIENTIFIC SERVICE COMMITTEE

The Scientific Service Committee of the Illinois State Medical Society has endeavored for many years to give all possible assistance to county medical societies in selecting speakers to appear for talks on any subject in medicine which they desire to have presented.

In order to give better service the Committee has prepared a new classified listing of scientific subjects on which physicians are prepared to speak. These listings will be of material aid to county society secretaries in arranging programs in post graduate education.

The Committee will welcome requests for service from the secretaries and complete information may be had by writing to Miss Jean McArthur, Secretary, Educational Committee, Illinois State Medical Society, 30 North Michigan Avenue, Chicago.

POWER GIVEN TO ONE MAN, NOT ELECTED, TO WORK AS HE PRESCRIBES

Merle Thorpe, in *Nation's Business*, under the heading "BUREAUCRAT BECOMES DICTOCRAT" says:

Now comes a round-about official admission of a condition we have described in these columns during the past decade, a condition at once sapping the freedom of the individual citizen and destroying business enterprise, which is so sorely needed.

The admission comes from Senator Logan, of Kentucky, and Congressman Walter, of Pennsylvania—ironically enough, from men who helped by their votes to bring about the situation they now recognize and hope to alleviate, not by repeal, but by another layer of legislation.

The Logan-Walter bill is most significant. It proposes to give a citizen the opportunity to present his case to the courts, to have a trial by a jury of his peers. It offers a free man relief from the dictocrat (in a bureau, board or commission) who has deprived him of his freedom or his property.

The dictocrat is a natural development of the bureaucrat. The earlier bureaus of the federal Government did foolish and wasteful things. They "researched" and printed millions of pamphlets on "The Love Life of the Bull Frog," and "How to Pin Diapers on the Baby." But these were harmless extravagances; they were not invested with the power to legislate, to judge, and to deny judicial appeal. That development has come in the past ten years with terrifying rapidity, with more than 50 executive agencies, employing tens of thousands of men and women, now exercising the powers of autocracy. The bureaucrat has become dictocrat.

From a thousand examples of this alien practice, new to America, but very, very old in Europe, the case of Mr. Andrews, wage-hour executive agent of Congress, is typical. He announces that he has changed his mind and that employees receiving $200 a month or more are no longer exempt from the Act. Here is a power given to one man, not elected, to compel citizens to work as he prescribes, to single out an individual or a group for punitive or favorable treatment. His whim or caprice may rule today, tomorrow he may be rewarding friends.

In this case there is not need of conjecture. Mr. Andrews slips in an admission that he is rewarding the labor union because "it has done such a swell job in helping me fight my battles."

A similar example of this departure from a government by written law and to a government by men is the Labor Department's instruction to federal employment agencies — public, tax-supported agencies — not to fill positions for those employers against whom there is a grievance pending, even though the grievance may be outside his plant, such as a jurisdictional dispute.

No business today, from steel maker to peanut vendor, has written sailing orders.

The law of the land is found not on statute books, but in the brief cases of a hundred thousand dictocrats.

The Logan-Walter bill confesses the disease but, instead of a prescription to eliminate the causes, simply adds another layer of law, shamefully admitting that a citizen today in this erstwhile land of the free, must, by a new law, be given the protection of the courts. A doubtful protection, indeed, when it is recalled that court reviews, of the Wagner Labor Board's decisions have cost the citizen who appealed an average of $26,000, and the sorry spectacle of re-enacting the trial by jury guarantee in the Bill of Rights.

Much can be said, and should be said, of the loss of personal freedom. But here we shall emphasize the point again, that one of the chief obstacles to a resumption of business enterprise is this new and un-American method of making the laws under which a business can carry on.

No matter how carefully a manager may sweat out an operating budget there is always a specter at his desk, the specter of a new rule that may wreck it completely. No skill, no vision can meet the additional risk of a ruling reversed overnight when a friendly group does "a swell job in fighting my battles."

Comment

As we have said and repeated frequently over two decades *"Bureaucracy Is Always a Curse and Where Medicine Is Involved It Is Fatal."* Now we have Dictatorship plus *Bureaucracy.*

MORTALITY UP—BIRTHS DOWN IN ILLINOIS

A release from the department of public health says:

Due chiefly to a substantial increase in the number of fatalities attributed to heart disease, deaths in Illinois from all causes during the first half of 1939 stood at 45,932 against 42,966 in that period of 1938, an unfavorable difference of 2,966. The number of births was 55,614 and 57,233 for the first half of this and last year,

respectively, a decline of 1,619. Excess of births over deaths was only 9,682 in the first half of this year against 14,267, last.

Heart disease, to which was attributed 14,321 and 12,610 deaths in the first half of this year and last, respectively, an increase of 1,711, was by a wide margin the principal factor, responsible for the higher mortality this year. Limited largely to persons in the upper age brackets, the rising death rate from heart disease can be no surprise to any student of public health or demography. With the control over communicable diseases and other hazards of young life growing constantly more successful and with a declining birth rate, the average age of the general population has increased sharply, bringing with it an inevitable increase in the death rate. If prevailing birth and immigration rates continue, the general death rate will undoubtedly continue to rise until it reaches 15 or 16 per 1,000 population and the chief causes of death will continue to be the chronic ailments of later life, heart disease, cancer, nephritis, apoplexy, diabetes etc., all of which have followed an upward trend this year.

The maternal death rate declined noticeably, 3.2 against 3.4 per 1,000 births while the infant death rate remained unchanged, 42.5 per 1,000 births. Sharply higher mortality was attributed to influenza and whooping cough, due to unusual epidemic prevalence, and slightly higher rates to pneumonia and tuberculosis. Otherwise the trend in mortality from communicable diseases was downward.

INTERNATIONAL MEDICAL ASSEMBLY INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA

October 30, 31, *November* 1, 2, *and* 3, 1939

Pre-assembly clinics October 28, *post-assembly clinics, November* 4—*Chicago Hospitals*

CHICAGO, ILLINOIS

Monday, October 30

8:00 A. M.

Diagnostic Clinic: "Nervous Indigestion." Dr. Walter C. Alvarez. Professor of Medicine. University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minnesota.

Diagnostic Clinic: "Low Back Pain with Sciatica." Dr. Philip Lewin, Associate Professor of Orthopedic Surgery, Northwestern University School of Medicine, Chicago, Illinois.

Diagnostic Clinic: "Clinical Types of Nephritis." Dr. Soma Weiss, Hersey Professor of the Theory and Practice of Physic, Harvard Medical School; Physician-in-Chief, Peter Bent Brigham Hospital, Boston, Massachusetts.

Intermission for Review of Exhibits.

Diagnostic Clinic: "Differential Diagnosis of Lesions of the Right Colon." Dr. Fred W. Rankin, Lexington, Kentucky.

Diagnostic Clinic: "An Evaluation of the Major Operations for Cavernous Pulmonary Tuberculosis." Dr. John Alexander Professor of Surgery, University of Michigan Medical School, Ann Arbor, Michigan.

NOON INTERMISSION

1:00 P. M.

Diagnostic Clinic: "The Use of the Bone Graft in the Treatment of Bone Tumors." Dr. Dallas B. Phemister, Professor of Surgery, University of Chicago School of Medicine, Chicago, Illinois.

Diagnostic Clinic: "The Treatment of Osteoarthritis." Dr. Russell L. Haden, Cleveland Clinic, Cleveland, Ohio.

Address: "Surgical Aspects of Peptic Ulcer." Dr. Eldridge L. Eliason, Professor of Surgery, University of Pennsylvania School of Medicine Philadelphia, Pennsylvania.

Address: "Symptoms and Diagnosis of Obscure Fever." Dr. James G. Carr, Professor of Medicine, Northwestern University School of Medicine, Chicago, Illinois.

Intermission for Review of Exhibits.

Address: "Surgical Treatment of Gallstones." Dr. Elliott C. Cutler, Moseley Professor of Surgery, Harvard Medical School, Boston, Massachusetts.

Address: "Medical Treatment of Liver Disease." Dr. Albert N. Snell, Professor of Medicine, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minnesota.

Address: "Infections of the Upper Urinary Tract." Dr. C. Donald Creevy, Assistant Dean and Associate Professor of Surgery and Urology, University of Minnesota Medical School, Minneapolis, Minnesota.

DINNER INTERMISSION

7:00 P. M.

Address: "Allergy as a Factor in General Medicine." Dr. James H. Black, Professor of Preventive Medicine, Baylor University College of Medicine, Dallas, Texas.

Address: "Management of Ileostomy and Colostomy." Dr. Richard B. Cattell, Lahey Clinic, Boston, Massachusetts.

Address: "Use of Hormones in Obstetrics." Dr. Frederick H. Falls, Professor of Obstetrics and Gynecology, University of Illinois College of Medicine, Chicago, Illinois.

Address: "Abdominal Pain in Extra-Abdominal Origin." Dr. John H. Musser, Professor of Medicine, Tulane University School of Medicine, New Orleans, Louisiana.

Address: "How Can High Mortality Rate from Skull Fractures Be Reduced?" Dr. Harry Mock, Associate Professor of Surgery, Northwestern University School of Medicine, Chicago, Illinois.

Address: "Treatment of Pellagra and Associated Deficiencies." Dr. Tom D. Spies, Associate Professor of Medicine, University of Cincinnati College of Medicine, Cincinnati, Ohio.

Tuesday, October 31

8:00 A. M.

Diagnostic Clinic: "Syphilis of the Vascular System." Dr. James E. Paullin, Professor of Clinical Medicine, Emory University School of Medicine, Atlanta, Georgia.

Diagnostic Clinic: "Fractures of the Femur Treated by Buck's Extension." Dr. William R. Cubbins, Professor of Bone and Joint Surgery, Loyola University School of Medicine, Chicago, Illinois.

Diagnostic Clinic: "Types of Edema and Their Treatment." Dr. Reginald Fitz, Wade Professor of Medicine, Boston University School of Medicine, Boston, Massachusetts.

Intermission for Review of Exhibits.

Diagnostic Clinic: "Treatment of Increased Intracranial Pressure." Dr. Eric Oldberg, Professor and Head of the Department of Neurology and Neurological Surgery, University of Illinois College of Medicine, Chicago, Illinois.

Diagnostic Clinic: "Management of Obesity." Dr. Robert W. Keeton, Professor of Medicine, University of Illinois School of Medicine, Chicago, Illinois.

NOON INTERMISSION

1:00 P. M.

Diagnostic Clinic: "Diagnosis and Treatment of Tumors of the Intestine." Dr. W. Wayne Babcock, Professor of Surgery and Clinical Surgery, Temple University School of Medicine, Philadelphia, Pennsylvania.

Diagnostic Clinic: "Diseases of the Lungs Simulating Tuberculosis." Dr. Charles R. Austrian, Associate Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Address: "Anomalies of the Lower Urinary Tract." Dr. Hugh H. Young, Professor of Urology, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Address: "The Physicians Interest in Gall-Bladder Disease." Dr. Alvah H. Gordon, Professor of Medicine, McGill University Faculty of Medicine, Montreal, Canada.

Intermission for Review of Exhibits.

Address: "Indications for Cesarean Section." Dr. Nicholson J. Eastman, Professor of Obstetrics, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Address: "Rupture of Intervertebral Disks as a Cause of Low Back Pain and Chronic Recurring Sciatica." Dr. Alfred W. Adson, Professor of Neurosurgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minnesota.

Address: "Cerotherapy in the Treatment of Pneumonia." Dr. Gerald S. Shibley, Associate Clinical Professor of Medicine, Western Reserve University School of Medicine, Cleveland, Ohio.

DINNER INTERMISSION

7:00 P. M.

Address: "Exophthalmos" (The Joseph Schneider Foundation Presentation). Dr. Albert D. Ruedemann, Cleveland Clinic, Cleveland, Ohio.

Address: "Medical Treatment of Peptic Ulcer." Dr. Ralph C. Brown, Clinical Professor of Medicine, Rush Medical College, Chicago, Illinois.

Address "The Management of Intestinal Obstruction." Dr. Thomas G. Orr, Professor of Surgery, University of Kansas School of Medicine, Kansas City, Missouri.

Address: "Migraine." Dr. Harold G. Wolff, Assistant Professor of Medicine, Cornell University Medical College, New York, New York.

Address: "Operability of Carcinoma of the Stomach." Dr. Verne C. Hunt, Clinical Professor of Surgery, University of Southern California School of Medicine, Los Angeles, California.

Address: "The Present Status of Transurethral Resection. Dr. Herman L. Kretschmer, Clinical Professor of Surgery (Genito-Urinary), Rush Medical College, Chicago, Illinois.

Wednesday, November 1

8:00 A. M.

Diagnostic Clinic: "Lesions of the Right Upper Quadrant of the Abdomen." Dr. William F. Rienhoff, Associate Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Diagnostic Clinic: "Rheumatic Fever in Children." Dr. Robert A. Black, Professor of Pediatrics, Loyola University School of Medicine, Chicago, Illinois.

Diagnostic Clinic: "Goitre and the Heart." Dr. Wallace M. Yater, Professor of Medicine and Director of the Department of Medicine, Georgetown University School of Medicine, Washington, D. C.

Intermission for Review of Exhibits

Diagnostic Clinic: "The Present Status of the Surgical Treatment of Peptic Ulcer." Dr. Howard K. Gray, Assistant Professor of Surgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minnesota.

Diagnostic Clinic: "Experiences with the Surgical Treatment of Hypertension." Dr. Loyal Davis, Professor of Surgery, Northwestern University School of Medicine, Chicago, Illinois.

NOON INTERMISSION

1:00 P. M.

Diagnostic Clinic: "The Management of the Various Types of Goitre." Dr. Frank H. Lahey, Lahey Clinic, Boston, Massachusetts.

Diagnostic Clinic: "The Insulins in the Treatment of Diabetes." Dr. Elliott P. Joslin, Clinical Professor of Medicine, Harvard Medical School, Boston, Massachusetts.

Address: "Treatment of Acute Spreading Peritonitis Following Ruptured Appendix." Dr. William D. Haggard, Professor of Surgery, Vanderbilt University School of Medicine, Nashville, Tennessee.

Address: "Newer Approach to the Etiology and Treatment of Angina Pectoris." Dr. William J. Kerr, Professor of Medicine, University of California Medical School, San Francisco, California.

Address: "Carcinoma of the Lung." Dr. Evarts A. Graham, Professor of Surgery, Washington University School of Medicine, St. Louis, Missouri.

Address: "Dysmenorrhea." Dr. John R. Fraser, Professor of Obstetrics and Gynecology, McGill University Faculty of Medicine, Montreal, Canada.

Address: "The Hormones of the Gastro-Intestinal Tract," Dr. Andrew C. Ivy, Nathan Smith Davis, Professor of Physiology and Professor of Pharmacology, Northwestern University School of Medicine, Chicago, Illinois.

ASSEMBLY DINNER

For members of the profession, their ladies and friends.
Informal

7:00 P. M.

Dr. George W. Crile, President of the Inter-State Postgraduate Medical Association of North America—Master of Ceremonies.

Addresses by:

Major General James C. Magee, Surgeon-General, United States Army, Washington, D. C.

Dr. James B. Herrick, Professor Emeritus of Medicine, Rush Medical College, University of Chicago, Chicago, Illinois.

Major General Ross T. McIntire, Surgeon-General, United States Navy, Washington, D. C.

Dr. Chevalier Jackson, Honorary Professor of Broncho-Esophagology, Temple University School of Medicine, Philadelphia, Pennsylvania. President-Elect Inter-State Postgraduate Medical Association of North America.

Dr. James H. Hutton, President, Illinois State Medical Society, Chicago, Illinois.

Dr. Nathan S. Davis III, Assistant Professor of Medicine, Northwestern University School of Medicine, Chicago, Illinois. President of Chicago Medical Society.

Thursday, November 2

8:00 A. M.

Diagnostic Clinic: "Malaria and Narcotic Addiction." Dr. Italo F. Volini, Professor of Medicine, Loyola University School of Medicine, Chicago, Illinois.

Diagnostic Clinic: "Joint Fractures." Dr. John J. Moorhead, Professor of Clinical Surgery, New York Postgraduate Medical School, New York, New York.

Diagnostic Clinic: "Modern Aspects of the Diagnosis and Management of Hypertension." Dr. Roy W. Scott, Professor of Clinical Medicine, Western Reserve University School of Medicine, Cleveland, Ohio.

Intermission for Review of Exhibits

Diagnostic Clinic: "Differential Diagnosis of Tumors of the Breast." Dr. John F. Erdmann, Attending Surgeon, New York Postgraduate Medical School, New York, New York.

Diagnostic Clinic: "Clinical Types of Pituitary Diseases." Dr. Walter Timme, Professor of Clinical Neurology, Columbia University College of Physicians and Surgeons, New York, New York.

NOON INTERMISSION

1:00 P. M.

Diagnostic Clinic: "A Presentation of Dermatological Cases." Dr. Francis E. Senear, Professor of Dermatology, University of Illinois College of Medicine, Chicago, Illinois.

Diagnostic Clinic: "Diagnosis and Treatment of Diseases of the Cranial Nerves." Dr. Walter E. Dandy, Adjunct Professor of Neurological Surgery, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Address: "Chemotherapy in the Treatment of Pneumonia." Dr. Russell L. Cecil, Professor of Clinical Medicine, Cornell University Medical College, New York, New York.

Address: "Clinical and Biological Problems of the Irradiation of the Ovary." Professor Dott, Emanuele Momigliano, Department of Obstetrics and Gynecology, Royal University of Rome, Rome, Italy.

Intermission for Review of Exhibits

Address: "The Clinical Use of Digitalis." Dr. Drew W. Luten, Associate Professor of Clinical Medicine, Washington University School of Medicine, St. Louis, Missouri.

Address: "Interrelation of the Organs of Internal Secretion." Dr. Elmer L. Sevringhaus, Professor of Medicine, University of Wisconsin Medical School, Madison, Wisconsin.

Address: "Vascular and Abdominal Surgery." Dr. Charles H. Phifer, Professor of Surgery, University of Illinois School of Medicine, Chicago, Illinois.

DINNER INTERMISSION

7:00 P. M.

Address: "Prevention and Treatment of Virus Disease." Dr. Thomas M. Rivers, Rockefeller Institute for Medical Research, New York, New York.

Address: "Stricture of the Common and Hepatic Ducts." Dr. Waltman Walters, Professor of Surgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minnesota.

Address and Movie: "Transplantation of the Ureters into the Recto-Sigmoid and Cystectomy for Malignant Tumor of the Bladder." Dr. William E. Lower, Cleveland Clinic, Cleveland, Ohio.

Address: "Management of the Menopause." Dr. Emil Novak, Associate in Gynecology, Johns Hopkins University Medical School, Baltimore, Maryland.

Address and Movie: "The Distended Colon; Its Medical and Surgical Management." Dr. Claud F. Dixon, Associate Professor of Surgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minnesota.

Address and Movie: "Fifty Years of Eclampsia, Placenta Previa, and Cesarean Section." Dr. Joseph B. DeLee, Professor Emeritus of Obstetrics and Gynecology, University of Chicago School of Medicine, Chicago, Illinois.

Friday, November 3

8:00 A. M.

Diagnostic Clinic: "Diagnosis of Diseases of the Gall-Bladder." Dr. Warren W. Cole, Professor of Surgery, University of Illinois School of Medicine, Chicago, Illinois.

Diagnostic Clinic: "Diet and Nephritis." Dr. James S. McLester, Professor of Medicine, University of Alabama School of Medicine, Birmingham, Alabama.

Diagnostic Clinic: "Diagnostic Significance of Pain in the Abdomen." Dr. Irvin Abell, Clinical Professor of Surgery, University of Louisville School of Medicine, Louisville, Kentucky.

Intermission for Review of Exhibits.

Diagnostic Clinic: "Use of Iron, Liver Extract and Desiccated Stomach in the Treatment of Anemia." Dr. Cyrus C. Sturgis, Professor of Internal Medicine, University of Michigan School of Medicine, Ann Arbor, Michigan.

Diagnostic Clinic: "Is Early Hypertension a Curable Disease?" Dr. George W. Crile, Cleveland Clinic, Cleveland, Ohio.

NOON INTERMISSION

1:00 P. M.

Diagnostic Clinic: "Diagnostic Features of Chronic Appendicitis." Dr. George P. Muller, Professor of Surgery, Jefferson Medical College, Philadelphia, Pennsylvania.

Diagnostic Clinic: "Treatment of Peripheral Vascular Diseases." Dr. Alton Ochsner, Professor of Surgery, Tulane University of Louisiana School of Medicine, New Orleans, Louisiana.

Address: "The Management and Treatment of Scarlet Fever." Dr. John A. Toomey, Associate Professor of Pediatrics, Western Reserve University School of Medicine, Cleveland, Ohio.

Address: "Diagnosis of Diseases of the Thyroid Gland." Dr. Donald Guthrie, Associate Professor of Surgery, University of Pennsylvania Graduate School of Medicine, Sayre, Pennsylvania.

Address: "The Jaundiced Bleeder: Control of Hemorrhage with Special Reference to Vitamin K. Therapy." Dr. Harry P. Smith, Professor of Pathology, State University of Iowa, Iowa City, Iowa.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next written examination and review of case histories (Part I) for Group B candidates will be held in various cities of the United States and Canada on Saturday, January 6, 1940, at 2:00 p. m. The Board announces that it will hold only one Group B, Part I, examination this year prior to the final general examination (Part II), instead of two as in former years. Candidates who successfully complete the Part I examination proceed automatically to the Part II examination held in June, 1940.

Applications for admission to Group B, Part I, examinations must be on file in the Secretary's office not later than October 4, 1939.

The general oral and pathological examinations (Part II) for all candidates (Groups A and B) will be conducted by the entire Board, meeting in Atlantic City, N. J., on June 8, 9, 10, and 11, 1940, immediately prior to the annual meeting of the American Medical Association in New York City.

Applications for admission to Group A, Part II examinations must be on file in the Secretary's office not later than March 15, 1940.

After January 1, 1942, there will be only one classification of candidates, and all will be required to take the Part I examinations (written paper and case records) and the Part II examinations (pathological and oral.)

For further information and application blanks, address Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh (6), Pennsylvania.

THIRD ANNUAL SYMPOSIUM

DEPARTMENT OF INDUSTRIAL MEDICINE

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

September 25 and 26, 1939

SCIENTIFIC SESSIONS

Monday Morning, September 25, 1939—9:00 A. M.

Chairman—Dr. Paul Starr, Assistant Professor of Medicine, Northwestern University Medical School

Non-Tuberculous Pulmonary Diseases

I. Clinical Aspects—Dr. Ernest E. Irons, Chairman

of the Department of Medicine, Rush Medical College.

II. Radiological Aspects—Dr. Hollis Potter, Chicago.

Discussion by—Dr. Robert C. Bloch, University of Chicago; Dr. Henry Sweany, Municipal Tuberculosis Sanitarium; Dr. E. B. Neff, Deere & Co.

Monday Afternoon, September 25, 1939—1:30 P. M.

Chairman—Dr. Wilbur E. Post, Clinical Professor of Medicine, Rush Medical College

Kidney Diseases of Mid Life

I. Clinical Aspects—Dr. Herman O. Mosenthal, Clinical Professor of Medicine, New York Post-Graduate Medical School of Columbia University.

II. Pathological Aspects—Dr. J. P. Simonds, Professor of Pathology, Northwestern University Medical School.

Discussion by—Dr. Edwin F. Hirsch, St. Luke's Hospital; Dr. Fred Fitz, Chicago Tribune; Dr. R. J. DeMotte, Pullman-Standard Car Mfg. Co.

Tuesday Morning, September 26, 1939—9:00 A. M.

Chairman—Dr. Edward Oliver, Associate Professor of Dermatology, Rush Medical College

Soap—A Cleanser and an Irritant and Its Role in Occupational Dermatitis

Dr. James Herbert Mitchell, Associate Professor of Dermatology, Rush Medical College.

Discussion by—Dr. L. F. Weber, University of Illinois Medical School; Dr. Cleveland White, Northwestern University Medical School; Dr. B. B. Reeve, Standard Oil Company; Dr. K. K. Jones, Northwestern University Medical School.

Welding—Gas and Electric

I. The Relative Importance of the Health Hazard—Dr. Eugene L. Walsh, Fellow in Medicine, Northwestern University Medical School.

II. The Protection of Operators—Dr. James A. Britton, Associate Professor of Medicine, Northwestern University Medical School.

Discussion by—Dr. Sanford Gifford, Northwestern University Medical School; Mr. Warren A. Cook, Zurich General Accident & Liability Ins. Co., Dr. H. A. Vonachen, Caterpillar Tractor Company.

Tuesday Afternoon—September 26, 1939—1:30 P. M.

Chairman—Dr. M. Herbert Barker, Assistant Professor of Medicine, Northwestern University Medical School

Common Beneficient Uses of Poisonous metals and Alkaloids—Economic Importance and Health Hazards

I. Lead and Lead Alloys in Industry—Dr. Marcus A. Grossmann, Director of Research, Carnegie Illinois Steel Corporation.

II. Control or Elimination of Health Hazards in Manufacture and Use of Ledloy—Dr. A. G. Kammer, Medical Director, Inland Steel Co.

III. Metallic Salts and Alkaloids in Agriculture.

IV. The Protection of the Health of the Grower, the Handler and the Consuming Public—Professor W. A. Ruth, Department of Horticulture, University of Illinois.

Discussion by—Dr. Frank Queen, Passavant Hospital.

BANQUET SESSION

Blackstone Hotel, 7:00 P. M., September 26, 1939

Chairman—Dr. J. H. Chivers, Chairman, Board of Governors, Department of Industrial Medicine, Northwestern University Medical School

Toastmaser—Dr. Irving S. Cutter, Dean, Northwestern University Medical School

THE PAST AND FUTURE OF PREVENTIVE MEDICINE

Dr. Walter L. Bierring

Commissioner, Iowa State Department of Health, Des Moines, Iowa

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OSTEOPATHY VETOED IN NEW YORK

On May 10 the governor of New Lork vetoed a bill which would have permitted licensed osteopaths to use instruments for minor surgical work, as well as use anesthetics, antiseptics, narcotics and biologic products. In a message explaining his action, the governor wrote:

To the Assembly:

There has been great misunderstanding with regard to this bill. Many people apparently believe that the practice of osteopathy as now carried on depends on my approval of the bill. This, of course, is completely contrary to the facts.

Any person now licensed or hereafter licensed as doctors of osteopathy are, regardless of my action on the bill, permitted to render exactly the same services as in the past. Their authority to carry on the functions now performed is in no way curtailed or abridged.

VALUE OF OSTEOPATHY CONCEDED

There is no question as to the substantial value and usefulness of osteopathy. This is generally conceded and recognized. This bill would, however, give all licensed osteopaths broad additional powers. It would permit all licensed osteopaths to use instruments for minor surgical procedures, to administer anesthetics and antiseptics, and to prescribe narcotics and biological products.

The additional authority now asked by the osteopaths may be far-reaching in its effect. A minor operation, if not properly performed, I am advised, may be more serious in its effect upon the patient than some of the so-called important operations.

The administering of drugs and biologicals where there is not sufficient training and experience with regard to their effect on the patients may lead to serious ill effects.

Undoubtedly many of the persons now licensed to practice osteopathy in this State have had broad training and experience in matters relating to medicine and surgery. On the other hand, many of the osteopaths practicing in this State were licensed prior to the

setting of the present high standards of training and have had little or no later experience in medicine or surgery.

LICENSING METHODS RECALLED

In addition, a very substantial number of the osteopaths now practicing in this State receive their licenses not through examinations within the State but by endorsement of licenses granted to them in other States. Some of them are graduates of osteopathic schools which are no longer recognized by our board.

In my opinion legislation should provide that applicants for licenses and those who already hold licenses as osteopaths but who now desire additional powers should be required to satisfy the Board of Regents either by submitted credentials or by examination that they have had the proper instruction and training in surgical procedure and drug therapy to justify the granting of the additional powers set forth in this bill.

In this way the Regents would be able to determine those who are actually qualified to use instruments for minor surgical procedures, to administer anesthetics and antiseptics and to prescribe narcotics and biological products. The difficulty of providing for such determination by the Board of Regents does not seem to me great.

The bill is disapproved.

CHRONIC STREPTOCOCCIC ULCER OF THE SKIN: UNRESPONSIVE TO LOCAL THERAPY BUT CURED BY SULFANILAMIDE: REPORT OF TWO CASES

The type of ulcer in the two cases that form the basis of M. H. Goodman's, Baltimore (Journal A. M. A., Oct. 15, 1938), report is apparently unique in that it was produced by streptococci, presented features of chronicity both clinically and histologically and occurred on the skin of healthy individuals. In each of the two cases there was introduced into the skin a variety of beta hemolytic streptococcus capable of producing low grade necrosis leading to chronic ulceration. The organism proved to be a Streptococcus pyogenes of human strain. In both cases the ulcers had enlarged with an irregular, firm, thickened, angrily red, ragged, undermined border and uneven deepening of a roughly granular glazed base, discharging continuously a copious serous fluid. Other than a slight and transient regional lymphadenitis, there were no complications. The biopsy studies revealed the presence of intracellular and extracellular cocci in pairs and short chains, and in various types of mediums inoculated with exudate from the ulcers a pure culture of the streptococcus was obtained. In case 1 it was totally impossible to effect healing by means of local antiseptic treatment. This was unquestionably due to the fact that the organisms, enclosed in a chronic fixed tissue cellular infiltrate in the region of the undermined portion of the ulcers, were beyond the reach of the agents applied. The sharp and prompt effect of sulfanilamide on the ulcer of the arm in this case not only solved the problem of therapy but gave confirmatory evidence that the ulcers were of primary streptococcus (beta hemolytic) etiology. The ulcers of the leg in case 2 were each about four times the size of the large ulcer of the right forearm in case 1. The effect of sulfanilamide on these ulcers was as sharp and prompt as in case 1. Sterilization of the lesions took place as a result of the initial "course" of sulfanilamide, ranging from a total daily dosage of 60 grains to the gradually reduced dose of three 5 grain tablets a day. The slower healing in case 2 as compared to that in case 1 was obviously due to the larger size of the ulcers, their location on the lower extremities and the presence of numerous varicose veins.

RECENT STUDIES ON PATHOGENESIS OF WERLHOF'S DISEASE

Mario Torrioli and Vittorio Puddu, Rome, Italy (Journal A. M. A., Oct. 15, 1938), state that the results of researches on Werlhof's disease quite recently carried out in the laboratories of Johns Hopkins Hospital appear to be in perfect unison with the ones made by Torrioli and his associates. By means of intravenous injections of a spleen digested extract from a patient with Werlhof's disease, Troland and Lee repeatedly obtained a strong diminution of the platelets in the circulating blood of rabbits. Troland and Lee seem to arrive at the conclusion that the thrombopenic substance is specific of the splenic tissue in Werlhof's disease. On this point, however, the authors wish to make a reservation. In the first place it should not be forgotten that a decrease of platelets in circulating blood was obtained by Torrioli and Pusic with normal spleen extract. In the second place the controls used by the American authors were thyroid tissue, myomatous uterus which, like muscular tissue, has almost no effect at all on the thrombopoietic system, and finally a spleen from a patient with Banti's disease and from one with hemolytic disorder, both of which differ widely from normal spleen as regards structural and functional plan. It is to be hoped that experiments with extracts of normal spleen and other normal organs will be made on a larger scale in order to set on a perfectly clear basis the point regarding the specific character of this principle, a point which appears to them of the utmost importance for the next step toward the future solution of the problem.

ARE TAXES HIGH?

Speaking editorially the *Daughters of America Magazine* for July says: "Government expenditures per capita are as follows: United States, $133; Great Britain, $123; France, $103. National expenditure for government is $523 per family. Politicians blame high taxes on relief work, whereas only $90 of the $523 represents state and federal relief. If none were on relief each family would still be taxed $433. With taxes in the United States the highest in the yorld, you have a fair indication of the present-day condition of American liberty."

Original Articles

ACUTE APPENDICITIS WITH PERFORATIVE PERITONITIS

KARL A. MEYER, M. D., PETER A. ROSI, M. D., ALFRED LUECK, M. D., and MALCOLM TODD, M. D.

CHICAGO

Acute appendicitis without perforation today offers no great surgical problem. Removal of the appendicitis, barring the few catastrophies, infections and pneumonic complications, is associated with a very low mortality. At the Cook County Hospital during the period of 1928-1932 there were 1,857 patients with acute appendicitis without perforation, with a mortality of 1.1 per cent., whereas, during the period of 1937-1938 there were 1,585 patients with acute nonperforative appendicitis, with five deaths or a mortality of 0.39 per cent. Individual surgeons have still lower mortality figures in their own series. Immediate appendectomy in view of our present knowledge is the best treatment of acute nonperforative appendicitis.

Acute appendicitis complicated by perforation of the appendix with local or diffuse peritonitis remains a serious surgical problem. Every year in the registration area of the United States approximately 16,000 deaths are reported from acute appendicular peritonitis. Every year the medical literature contains many articles devoted to acute appendicitis and its complications. Black reviewed 37 such articles, which contained reports on 83,144 cases operated upon by 1,500 surgeons in 150 hospitals. The mortality rate for the entire series was 4.55 per cent. with variations of 0.036 per cent. to 11.40 per cent. The mortality for acute perforative appendicitis complicated by diffuse peritonitis varied from 11.25 per cent. to 33.00 per cent. or an average of 24.19 per cent. This series is sufficiently large to give a fairly accurate picture of the results obtained in the treatment of acute appendicitis and its complications.

Important problems in the treatment of acute perforative appendicitis are prophylaxis and early surgical consultation. The patient's neglect in seeking medical advice for the early symptoms of acute appendicitis is probably the greatest contributing factor in allowing the appendix to perforate. Much needs to be done in educating the public regarding the classical symptoms of acute appendicitis and the seriousness of allowing the appendix to perforate. Self-medication, especially as pertaining to catharsis for indefinite abdominal distress continues to be an important factor in the mortality rate of perforative appendicitis. Cathartics are responsible not only for a greater incidence of perforations but to a more diffuse peritonitis. Upon the family physician falls the greatest responsibility of educating the public concerning these contributing factors. Upon druggists falls the responsibility of advising purchasers of cathartics to consult their family physician before trying purgaton for abdominal pain.

Present day medical practice rarely is responble for allowing the appendix to perforate. However, on account of the protean manifestation of acute appendicitis errors in diagnosis are not infrequent and occasionally an appendicular peritonitis occurs while the patient is under unsuspecting medical management. In view of the enormous differences in the mortality rate of acute appendicitis and of acute perforative appendicitis, exploration of the appendix of those patients in whom an acute appendicitis cannot be ruled out is justified.

Until the millennium is reached in which all the patients with acute appendicitis are operated upon before perforation occurs, the management of acute appendicular peritonitis shall continue to be one of surgery's greatest problems. In order to evaluate the various factors contributing to the mortality rate at the Cook County Hospital we reviewed 1,000 records of patients with acute perforative appendicitis during the period of 1928-1932 and subsequently reviewed the records of similar cases admitted in 1937 and 1938. As a basis for study we divided the patients into two groups: (1) acute perforative appendicitis with diffuse peritonitis, and, (2) acute perforative appendicitis with localizing peritonitis.

During the period of 1928 to 1932 there were 519 cases of acute appendicitis with perforation and diffuse peritonitis with 139 deaths or a mortality rate of 26.4 per cent. During the period of 1937 and 1938 there were 262 cases with 61 deaths, or a mortality rate of 23.3 per cent. These

From the Cook County Hospital, Chicago, Illinois.

Presented before Section on Surgery, 99th Annual Meeting, Illinois State Medical Society, Rockford, May 3, 1939.

mortality figures are higher than those reported by individual surgeons. However, in our series are included all cases admitted to the hospital. Some of the patients were moribund on admission, so that any treatment was futile. These figures, however, do give a picture of the diffuse

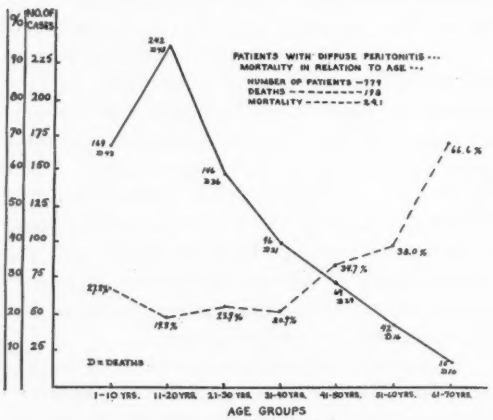

Fig. 1.

type of appendicular peritonitis seen in the indigent population. The mortality rate for the two periods was essentially the same. The 3.1 per cent. decrease in the death rate in the 1937-1938 period is probably statistically insignificant or might have been due to the more liberal use of blood transfusions made possible by our recently instituted blood bank and to the routine use of constant duodenal suction. The general principles of treatment were the same in both groups.

A review of the records of the two groups showed the relationship of age, duration of illness, type of operation and drainage of the peritoneal cavity to the mortality rate to be essentially the same so that the two series were considered together. The incidence of diffuse appendicular peritonitis and mortality rate in relation to age is shown in Fig. 1. The greatest number of patients, 242, or 31 per cent. of the total were between 11 and 20 years. There were 169 patients between one and ten years and 146 between 21 and 30 years.

In the remaining decades there was a proportionately decreasing incidence of diffuse peritonitis. The mortality rate in relation to age remained between 19.8 per cent. to 27.2 per cent. for the first four decades. There was a gradual rise in the fifth and sixth decades and an abrupt rise after 60 years of age. The slightly higher mortality in children under ten years is probably due to the fact that at this age the omentum is proportionately less developed than in adults, so that perforation of the appendix tends to produce a greater amount of leakage and a more rapid diffusion of the peritoneal infection than in adults. The gradual rise after 40 years is due in some degree to the diminished resistance, consistent with advancing years. However, in this group of cases, there was a greater number of errors in the diagnosis of acute appendicitis or of the ensuing appendicular peritonitis. In these older patients the symptoms were more atypical than in the younger groups and were more frequently mistaken for an acute nonsurgical abdominal infection. Misapplied conservative management in many of these cases was a contributing factor to the rising mortality rate. The highest mortality rate occurred after 60 years. In our series the incidence of diagnostic errors and postoperative complications was the greatest in this period.

The relationship of the duration of the disease to the mortality rate is illustrated in Fig. 2. The majority of patients, 299 or 40.4 per cent. of the entire group were operated upon during the second day of the disease; 195 patients or 26.3 per cent. were operated upon during the third day and 105 or 14.2 per cent. during the first twenty-four hours. There were only 66 cases or 8.9

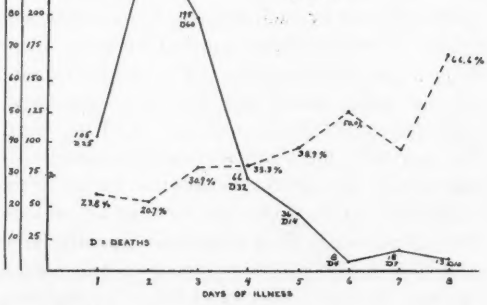

Fig. 2.

per cent. operated upon during the third day of the attack, the remaining patients were operated upon during the fifth to the eighth day of the disease. The mortality rate in relation to the duration of the disease remains fairly constant, 20.7 per cent. to 23.8 per cent., during the first

two days of the illness. After the second day the number of deaths gradually increases from 30.9 per cent. in the third day to 50 per cent. in the sixth and 66.6 per cent. in the eighth day. The rising mortality rate with the duration of the disease is consistent with the greater diffusion of the infections from a persistently leaking appendix. The peritoneal cavity may withstand the initial spilling from a perforated appendix but may be unable to withstand the continuous contamination for several days.

The relationship of the type of incision to the mortality rate is shown in Fig. 3. There were 426 patients who were operated upon through a McBurney incision with 82 deaths or a mortality rate of 19.2 per cnt. Whereas, there were 180 cases operated upon through a right rectus incision with 42 deaths or a mortality rate of 23.3 per cent. There were 11 patients upon whom a mid-line incision was made with five deaths or a mortality rate of 45.4 per cent. From these figures one can assume a gradually increasing mortality as the incisions are placed more medially. The ideal incision for an appendectomy in patients with a diffuse peritonitis is an incision that allows the greatest exposure of the appendix with a minimum exposure of the remaining peritoneal cavity. In this way the appendix can be removed as a source of infection without disturbing the localizing processes already in progress. The McBurney incision fulfills these qualifications. The McBurney in-

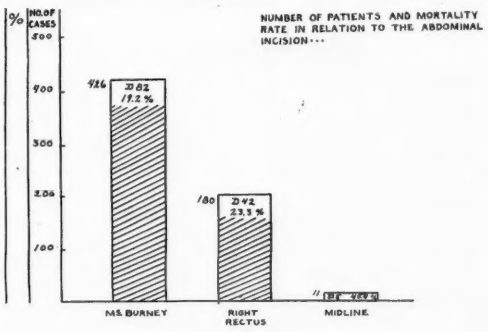

Fig. 3.

cision, especially if placed laterally near the anterior-superior spine of the ileum and with the middle of the incision over the maximum point of tenderness, has the advantage of entering the peritoneal cavity lateral to the cecum and usually directly over the leaking appendix. There is less manipulation of bowel or packing off of intestine than in the more medial incisions. If the appendix is retrocecal the McBurney incision can be enlarged toward the lumbar region, thereby freely exposing the retrocecal space. With the cecum and ascending colon dis-

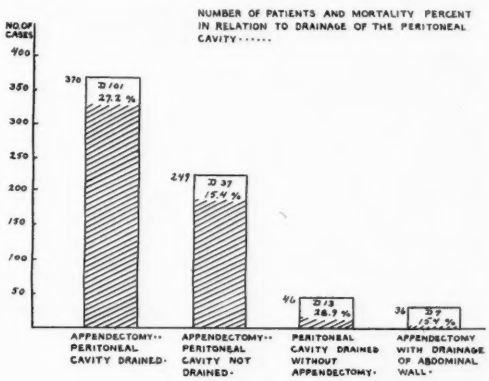

Fig. 4.

placed medially in these cases there is very little manipulation in the general peritoneal cavity. If the appendix lies medially or in the pelvis, the McBurney incision can be enlarged downward either by cutting across the internal oblique and transversalis muscles or cutting downward through the rectus fascia along the lateral border of the rectus muscle. Incisions placed more medially expose a greater extent of the peritoneal cavity and often interfere with the natural defensive mechanism that is attempting to wall off the perforated appendix. The mid-line incisions were made because of the errors in diagnosis.

Drainage of the peritoneal cavity continues to be a controversial procedure in the treatment of acute perforative appendicitis with diffuse peritonitis. As shown in Fig. 4 there were 370 patients upon whom an appendectomy was done and peritoneal cavity drained with 101 deaths or a mortality rate of 27.2 per cent. Whereas, there were 276 patients in whom appendectomy was done without drainage of the peritoneal cavity with 47 deaths or a 15.9 per cent. mortality. It would appear from these figures that drainage of the peritoneal cavity instead of decreasing the mortality rate actually increases the number of deaths. These groups figures may not be a just evaluation of the problem since some surgeons established drainage in the more

severely ill patients whereas they failed to drain the peritoneal cavity of the patients in moderately good condition. However, one can conclude that drainage or non-drainage of the peritoneal cavity probably are no factors in determining the eventual outcome of a patient with diffuse

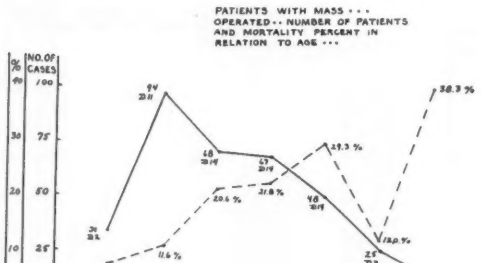

Fig. 5.

peritonitis. Drainage of the peritoneal cavity as shown experimentally by Gage is impossible for more than eight to 12 hours, at which time the drain is surrounded by fibrinous exudate and bowel which wall it off from the general peritoneal cavity. Necropsy studies likewise show that drains which are inserted at operation are walled off from the general peritoneal cavity by omentum, fibrinous exudate and adherent intestine. Statistical studies fail to show any pronounced reduction in the mortality that can be attributed to drains. The patients live or die, not because of the number of drains in their peritoneal cavity or the absence of such drains, but because of the extent of the diffuse peritonitis and the type of surgery that is done.

Much more important than drainage of the peritoneal cavity is the care of the abdominal wound following an appendectomy for a diffuse appendicular peritonitis. Such wounds are invariably contaminated by the purulent peritoneal exudate. Most of these wounds become infected postoperatively. The infection usually causes only local sloughing from which the patient recovers with little morbidity or at most an incisional hernia. We have had, however, four deaths from abdominal wall infections following removal of a perforated appendix. Recently we have scrubbed these wounds with soap and water, as described by Jackson, cut away any grossly infected fat, and closed the wound primarily with a small soft rubber tissue drain down through the muscles. Many of these wounds became infected but the infection was less severe than wounds that were not cleansed. An alternative method is to leave the incision open after the operation, only closing the peritoneal cavity. The abdominal wound is allowed to heal by secondary intention.

The postoperative treatment that gave the best results was a modification of the Ochsner regime. Fluids were given by intravenous drip; nothing was given by mouth or rectally. Blood transfusions were given liberally and continuous duodenal suction was used routinely. After the peritoneal infection was controlled and peristalsis was reestablished fluids were given orally and later followed by more solid foods. No large enemas were given because of the danger of "blowing out" the appendiceal stump, although small oil or water enemas were given as desired.

The second group of patients studied were those patients who had a mass, which was found on routine examination, palpated under anesthesia or found after the abdomen was opened. The presence of a mass was considered as an indication that the inflammatory process was being localized. In those patients who were operated upon, a perforated appendix was found in the center of the mass. Around the perforation there was a variable amount of pus. Forming the wall of this abscess there was a zone of fibrinous exudate, adherent intestines and omentum which protected the general peritoneal cavity from the inflammatory process. For

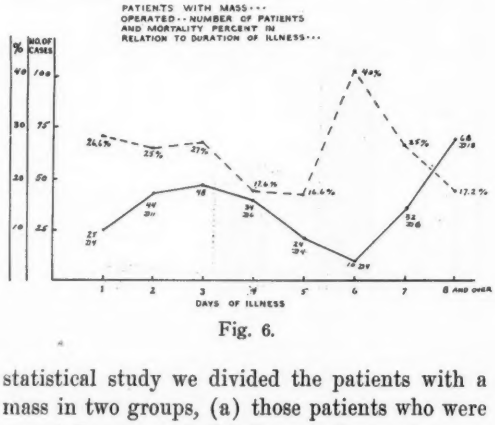
PATIENTS WITH MASS · · ·
OPERATED · · NUMBER OF PATIENTS
AND MORTALITY PERCENT IN
RELATION TO DURATION OF ILLNESS · · ·
NO. OF CASES
DAYS OF ILLNESS

Fig. 6.

statistical study we divided the patients with a mass in two groups, (a) those patients who were operated upon, and (b) those patients who were treated by the Ochsner management. Like in the treatment of diffuse peritonitis, the results during the 1928-1932 period and the 1937-1938

period were practically the same so that they were tabulated together.

There were 346 patients with a periappendicular mass who were operated upon, with 63 deaths or a mortality of 18.0 per cent. The incidence and mortality rate in relation to age is shown in Fig. 5. The highest incidence was between 11 and 40 years, after which the number of patients gradually decreased. The death rate was lowest between one and ten years and gradually increased until it reached 38.3 per cent. in patients past 61 years. This increasing mortality with advancing years, which was also present in patients with diffuse appendicular peritonitis, is due to the gradually diminishing resistance and the increasing incidence of diagnostic errors in the aged.

The number of patients and mortality rate in relation to the duration of the illness is shown in Fig. 6. The number of patients was greatest in the second and third days of the illness. Unlike in diffuse peritonitis where the incidence was greatest during the second day, in patients with a mass the incidence was evenly distributed throughout the eight-day period. The number of deaths was likewise more evenly distributed and decreased with the duration of the illness, whereas in diffuse peritonitis the deaths proportionately increased with the duration of the infection. The mortality rate was higher in those cases operated upon in the first three days and subsequently gradually decreased. The 40

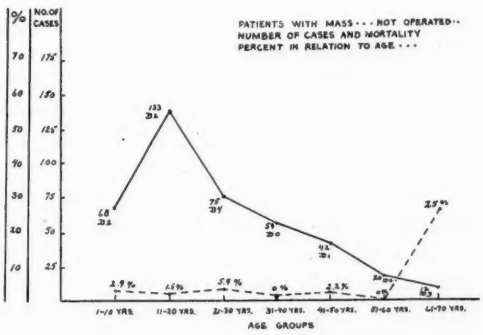

Fig. 7.

per cent. mortality in the sixth day is probably of no statistical importance on account of the small number of patients recorded. This decreasing mortality rate with the duration of the illness in patients with a periappendicular mass is directly the opposite result obtained in the analysis of patients with diffuse peritonitis in whom the mortality increased proportionately with the duration of the peritonitis.

In the group of patients with a periappendicular mass who were not operated upon there were 284 cases with 12 deaths or a mortality of 4.2

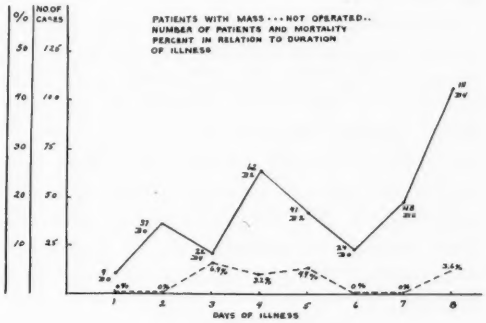
%
NO. OF CASES
PATIENTS WITH MASS · · · NOT OPERATED · ·
NUMBER OF PATIENTS AND MORTALITY
PERCENT IN RELATION TO DURATION
OF ILLNESS
DAYS OF ILLNESS

Fig. 8.

per cent. In Fig. 7 is illustrated the incidence and mortality rate according to age. The incidence curve has the same form as the curve of the patients with a mass who were operated upon. The number of patients is greatest between the age of 11 and 20 years. The mortality rate is very low in all age groups except in patients over 61 years, in whom the death rate rose abruptly to 25 per cent. However, after 61 years the number of patients is too small for statistical analysis.

Fig. 8 illustrates the number of patients and mortality per cent. in relation to the duration of the illness. The incidence is more or less evenly distributed over the recorded eight-day period. The mortality rate is low and evenly distributed throughout the entire period.

A review of the patients with a mass shows that the mortality is about four times as great in those patients operated upon (18 per cent.) as in the group not operated upon (4.2 per cent.). This mortality ratio was the same in the 1928-1932 and 1937-1938 groups.

During 1937-1938, however, there were proportionately more patients with a mass treated according to Ochsner's management than in the previous period. Because of this fact the mortality for the entire group of patients with a mass, operated and not operated upon combined, was lower. In 1928-1932 there were 481 patients with a mass with 59 deaths or a mortality of 12.2 per cent., whereas in 1937-1938 there were

249 patients with 16 deaths, or a mortality of 6.8 per cent., or a decrease in about 45.3 per cent. This reduced mortality rate for the group of patients with a mass was the only improvement in the treatment of the perforated appendix noted (1937-1938) over the previous period of 1928-1932.

Because of this reduced mortality and the reduction in the mortality of the nonperforative appendix from 1.1 per cent. in 1928-1932 to 0.3 per cent. in 1937-1938, the hospital mortality for all patients admitted with acute appendicitis was likewise lowered. During 1928-1932 there were 2,857 patients with 219 deaths, or a mortality rate of 7.6 per cent., during 1937-1938 there were 2,094 patients with 82 deaths or a mortality of 3.9 per cent., a 48.3 per cent., decrease.

SUMMARY

The two fundamental principles in treatment of acute perforative appendicitis are: (1) removal of appendix which nature has failed to "wall off" by a mass of fibrinous exudate, bowel and omentum from the general peritoneal cavity; and (2) assistance of the general reparative and immunological processes in those patients in whom there is evidence of localization or "walling off" of the peritonitis. The first would entail immediate appendectomy; the second delayed operation or the Ochsner treatment. The surgical therapy therefore depends upon the presence or absence of localization of the appendicular peritonitis. Many arbitrary rules, particularly in relation to the duration of the illness, have been established as guides to the most opportune time of operating upon these patients with appendicular peritonitis. But, since there are many variable factors following perforation of the appendix, rules often lead to meddlesome surgery or, not infrequently, to unnecessarily delayed surgery. There is no disease in which individualization of therapy is as important as in the treatment of diffuse appendicular peritonitis. There is also no disease in which the errors of commission are often as serious as the errors of omission. In the presence of definite evidence of localization such as a mass or gradually receding diffuse abdominal tenderness immediate surgery, as shown by the study of our figures increases the mortality rate. Since the appendix is being "walled off" from the peritoneal cavity the indications are to aid the localizing and immunologic processes. Ochsner's management which consists essentially of starvation and constant duodenal suction so as to put the bowel at rest and the administration of parentereal fluids, is the most physiologic procedure to follow. Surgical intervention in these cases may break through the protective barriers and convert a localized peritonitis into a diffuse peritonitis, or into what Bowers most aptly calls "induced spreading peritonitis." In our series we have had 12 patients with a mass, who were unquestionably recovering from perforated appendix with a localized peritonitis, die from diffuse peritonitis after untimely surgery. We have likewise reviewed many records of patients who were convalescing satisfactorily from an appendiceal mass or localized peritonitis and who became gravely ill with signs of spreading peritonitis after thoughtless surgery. Fortunately, most of these patients had enough resistance to overcome the operatively-induced spreading peritonitis. It has been our practice to treat all patients with a mass or localizing peritonitis by Ochsner management. Most of the patients will recover completely on this management alone. In a small percentage of patients the mass will become larger, the temperature and leucocyte count will begin to rise. In these patients who fail to respond satisfactorily to the Ochsner treatment the mass usually reaches sufficient size so that it comes in contact with the parietal peritoneum of the anterior abdominal wall, or bulges in the right lumbar region in case of a retrocecal appendix or into the rectum. An incision over the most prominent portion of the abscess will allow adequate drainage. Since the incision is within the area of adhesion between parietal peritoneum and abscess wall there will be no soiling of the peritoneal cavity by the pus draining from the abscess. Not infrequently an abdomen is opened under the mistaken diagnosis of acute appendicitis without perforation and a mass is found in the appendiceal area. But the mass is not adherent to the parietal peritoneum or is far removed from the abdominal wound so that an incision into the abscess will allow pus to drain into the peritoneal cavity and induce a spreading peritonitis. In these patients the abdominal wound is closed without attempting to drain the appendiceal mass. The patient is placed on Ochsner's management. In the majority of the patients

the mass will disappear. In a few instances the mass will increase in size and can be drained after it bulges in the right lower quadrant.

An alternative method of managing this type of appendiceal mass is to close the original incision and make a second incision directly over the mass. The abscess that is adherent to the parietal peritoneum may then be drained without soiling the peritoneal cavity. A third method of dealing with this problem is that if the abscess wall is not adherent to the perietal peritoneum the mass may be surrounded by iodoform gauze inserted between the mass and the parietal peritoneum so as to stimulate plastic exudate and adhesions around the abscess and between it and the abdominal wall. Twenty-four hours following this procedure the abscess may be safely drained.

The management of acute diffuse appendicular peritonitis continues to remain a difficult problem. The fundamental principle involved is whether or not the patient is suffering from a constantly leaking appendix, from which both appendiceal and cecal contents are continuously spilled into the peritoneal cavity, or, whether the patient has a peritonitis from the initial spilling of the appendiceal contents, the appendix in the meantime having been "walled off" by fibrinous exudate and is no longer a focus of infection to the peritoneal cavity. The clinical determination of these underlying pathologic processes is difficult and often impossible. The presence of a diminishing area of abdominal tenderness and its localization to the right lower quadrant is a valuable sign in determining cessation of leakage from the appendix or at least a "walling off" of the process to the periappendicular area. The indication of treatment in these patients is to assist the localization and build up the general immunological processes. The conservative Ochsner management will give a lower mortality rate in these patients than surgical intervention.

In the absence of definite findings of localization expeditious removal of the appendix is the best plan to follow, since the appendix is probably still a source of infection to the diffuse peritonitis. Surgery in these gravely ill patients should be limited to the region of the appendix and should be done with a minimum of trauma or manipulation of the intestines. A laterally placed McBurney incision with the middle of the incision over the point of greatest tenderness has given us the best results. It probably makes very little difference in the eventual mortality whether or not the peritoneal cavity is drained. Close observation during the postoperative period for signs and symptoms of complications and sequellae and the early treatment of these conditions will be helpful in reducing the mortality and make for a more uneventful convalescence. Meddlesome postoperative treatment that has no definite proven therapeutic effect should be avoided. Particularly dangerous are large enemas to combat abdominal distention. The danger of causing a "blow out" of the appendiceal stump far outweighs any good that can be obtained by giving enemas into a paralyzed bowel. Rigid adherence to the principle of starvation and constant duodenal suction so as to put the bowel at rest and intravenous administration of fluids, minerals and dextrose, will give the smoother postoperative course and lower mortality. A factor that has received too little attention in the past is the development of immunity to the organisms causing the diffuse peritonitis. Bower and his coworkers have shown a high titer to the antitoxin of the bacillus Welchii in a high percentage of patients recovering from appendicular peritonitis. He has likewise reduced the mortality rate from diffuse peritonitis by the administration of the Welch antiserum and convalescent serum. In view of the persistently high mortality with our present therapeutic procedures elucidation of the immunologic responses in patients with diffuse peritonitis may offer a new aid in the reduction of the mortality from acute perforative appendicitis.

DISCUSSION

Dr. Marshall Davison, Chicago: I think that Dr. Meyer is very modest in the presentation of his paper, particularly since he has used the editorial "we" and has never spoken in the first person. One might think the statistics he has presented are general, but in a period of 20 years of listening to Dr. Meyer as head of the Cook County Hospital we must realize that these statistics are the result of his teaching. For many years Dr. Meyer has had several points that he has brought before the interns; first, early operation for appendicitis, and second, the conservative management of patients who were seen late. I think those two points are the most important ones in the management of acute appendicitis. There is no point in discussing the management of an uncomplicated acute appendicitis. We are all agreed that it is a surgical lesion as soon as the diagnosis is made when that

diagnosis can be made early. The difficulties in management come from the complications of appendicitis, and not from the appendicitis alone. You have all heard the old saying that no one dies from appendicitis but that they die from the complications.

Dr. Meyer has very well divided these complications into the spreading type of periappendicitis, and the localized type of peri-appendicitis. If I may I would like to speak from the point of view of the appendiceal phlegmon and abscess. To be able to manage one of these cases intelligently the case must be seen for the first time late. We see many of these cases at the Cook County Hospital because most of them come under our management the second or third day. If we had seen them in the first twenty-four hours they would never have reached the stage of complications. I would say the majority come in with signs of localizing peritonitis, at which time we believe it is most dangerous to operate. Dr. Meyer has said the danger of producing peritonitis merely from the trauma of surgery is a factor that is not appreciated.

One might compare the formation of an appendiceal abscess to the formation of an ordinary boil. It goes through the stage of induration before the formation of a so-called head. We have all been taught since early days in pathology that the worst thing to do with a boil is to open it too early or squeeze it. We can say practically the same of an early appendiceal mass. These are not abscesses but are phlefmons comparable in that stage of development to the so-called boil. The mass may progressively enlarge for three, four, five or six days, and then much to our surprise in anywhere from 60 to 70 per cent. will subside spontaneously and completely disappear without any surgical intervention. Such masses in the early stage may have had small quantities of pus, but the resistance of the individual has been great and the pus is absorbed. Those that appear early and never reach the stage of suppuration are often continuously in the phlegmonous stage and will absorb much more rapidly than the larger masses which contain small amounts of pus. Dr. Meyer has spoken of the danger of opening these masses and has brought out the wisdom of closing the abdomen that is opened and found to contain one of such inflammatory masses, either with or without purulent exudate. Sixty or 70 per cent. of these masses will absorb without operative interference. Those that will require operative interference are those that have gone beyond the stage of induration and have developed into a well walled-off localized appendiceal abscess. Then surgical treatment is not undertaken with the idea of doing an appendectomy, but merely of draining a localized accumulation of pus within the abdomen.

I do not think there is a great deal of question but that every case of spreading appendicitis due to a leaking appendix should be operated upon early. I agree with Dr. Meyer that drainage does not make much difference in the ultimate mortality, because I do not believe it is possible to drain the entire abdominal cavity through a McBurney, right rectus or mid-line incision.

The most difficult time in appendicitis is in telling whether an abscess is going to localize; there are no symptoms or signs or anything in the history that will give information that the attack will subside and be treated as a localized appendiceal phlegmon. I really believe that probably the highest mortality in cases of appendicitis are in those which are treated conservatively but which should have been treated from the first with early appendectomy. Individuals in which pathological secretions localize following an appendiceal leak are fortunate. There are some in which localization begins at once. The first of the causes of localization is the anatomical position of the appendix; the retrocecal space is the most outstanding illustration. Second in the cause of localization is the presence of previously formed adhesions. We should say that the patient who has had previous attacks may develop a localized affair due to the resultant formation of adhesions. Third, thickness of the appendiceal wall; in this type the appendix will leak more slowly and this should presumably give nature more time to localize the process. Taking into consideration these three factors you will see that there is nothing by which we can tell at the onest of the attack whether they are present, or if present, whether they will work. Consequently we should not wait for localization in the early cases; we see localization under our eyes only at the time we see the patient for the first time late.

Dr. V. M. Seron, Joliet: I would like to ask Dr. Meyer two questions. First, in an acute ruptured appendix with beginning peritonitis, probably localized, which has been operated upon but the wound breaks open from pus drainage, how soon would he re-operate to close up at least the fasical and superficial layers, if not the peritoneum, where the bowel is pushing up into the wound.

Second, in a ruptured appendix with pus drainage what is the danger of intestinal obstruction after the wound has healed and adhesions have formed?

Dr. Frederick Christopher, Evanston: There is one point that Dr. Meyer did not mention which I think probably was not included in the subject matter of his talk; that it is good practice to advise everyone who has had an appendiceal abscess, who may have recovered spontaneously, to have a subsequent appendectomy.

Dr. A. D. Kirby, Champaign: How often in these cases of acute appendicitis do you find it necessary to do an enterostomy?

Dr. Karl Meyer, Chicago (closing): The secondary closure of an abdominal wound depends on the bacteriological examination. If there is still evidence of infection by culture, no secondary closure should be done. In answer to Dr. Christopher's question of when to operate following infection of the peritoneal cavity, it is our policy to wait for a period of at least two months. For a very large abscess, a longer time should ensue before the removal of the appendix is attempted. It is our policy to advise patients who have had a periappendiceal infection to have an appendectomy as only ten per cent. destroy themselves by infection or gangrene. Relative to the question of

enterostomy, it has been my belief and experience that it has only a very limited use in the treatment of paralytic ileus. The treatment of paralytic ileus is very important and demands very early care. The use of the Levine tube, adequate fluid balance, blood transfusions and medicinal agents for the stimulation of the intestinal musculature will be of great benefit to these patients. The question of mechanical ileus is a very important one and it occurs in a definite number of patients who are recovering from acute appendicitis. Here again it is of great importance to differentiate mechanical obstruction that demands reentering the abdominal cavity and one that is simply due to a paralytic ileus.

TUBERCULOUS ENTEROCOLITIS; DIAGNOSTIC DATA

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Rarely is one called upon to diagnose primary tuberculous enterocolitis. This condition is today a rarity, because its former prolific source, i. e., milk from tuberculous cows, has been almost entirely eliminated. The secondary type of tuberculous enterocolitis, on the whole, offers few diagnostic difficulties, since it is, almost invariably, preceded by pulmonary tuberculosis, which at this stage is most likely to be advanced. Therefore the occurrence of any gastro-intestinal disorder should then lead to the suspicion of, and inquiry into, the possible presence of tuberculous enterocolitis.

While it is generally held that there are no symptoms conclusively diagnostic of tuberculous enteritis, yet the mode of their onset and development, their inevitable effect on the body economy, together with the physical findings, x-ray and laboratory examinations, render a diagnosis fairly certain. It is the summation of all the symptoms, physical signs and laboratory data, rather than any particular ones, that determines the diagnosis.

The various symptoms occurring in intestinal tuberculosis have been reported from time to time by many authors, among them Brown,[1] Engelsmann,[2] Lemon,[3, 4, 5] and Logan.[6] These authors are very much in accord in their descriptions of the variable symptom-complex in intestinal tuberculosis. In the roentgen diagnosis of tuberculosis, however, there seems to be some difference of opinion as to the technique used and the interpretation of the findings. Due to the physiology and anatomy of the intestinal tract, no roentgen method has yet been devised which will definitely visualize areas of ulceration. The interpretations are based largely on the physio-

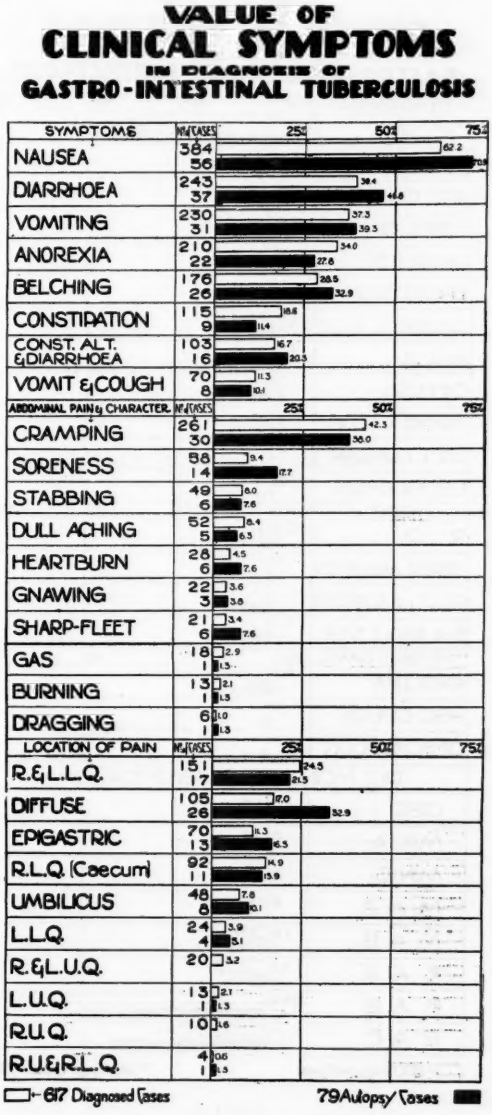

Chart 1.

Read before Section on Medicine, Illinois State Medical Society, Rockford, May 2, 1939.

logical changes that take place in the intestinal tract.

Stierlin[7] was among the first to recognize pathological changes in the lower end of the ileum, as well as indurating and ulcerative processes in the wall of the cecum and ascending

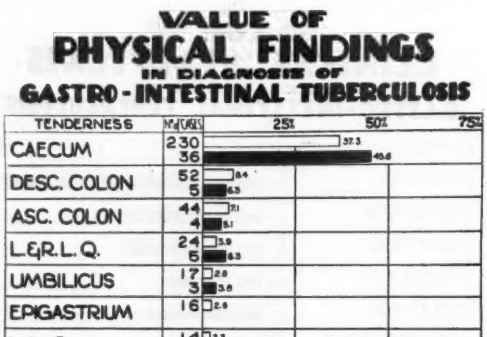

Chart 2.

colon. He summarizes his findings as follows: "The diagnosis of a stricture at the lower end of the ileum can be established by means of a radiogram which is taken five or six hours after an ingestion of the bismuth emulsion. Infiltrating, indurating, as well as ulcerative processes of the cecum and of the ascending colon manifest themselves regularly by an absence of the physiological shadow in this portion of the colon after five or six hours. For initial as well as advanced stages of the so-called cecal tuberculosis, the absence in the skiagram of the shadow of the cecum or of the cecum-ascending colon shadow is therefore typical. With the aid of radiography, the diagnosis can therefore be established even in cases in which it cannot be established clinically. Ulcers and infiltrations in the remaining portions of the colon can also be recognized in the skiagram by a shadow-defect which corresponds to the diseased section."

Brown and Sampson[8] confirm Stierlin's work, and in their conclusions state: "The presence of a spasm or spastic filling defects, irregular contour, lack of haustration, failure of the cecum or other parts of the proximal colon to retain barium, generalized hypermotility with complete or almost complete emptying of the colon in twenty-four hours, of confirmed segmentation with the dilatation of some coils of the small bowel, ileac stasis and gastric retention are the essential points in the roentgen diagnosis when the intestine is studied at the seventh, eighth, ninth or tenth hours and again at the twenty-fourth hour. The barium enema usually confirms the fact that the ulcerated cecum or other portions of the colon may fail to receive or to retain the barium. And, conversely, the normal colon usually is represented by smooth, even haustrations without decided irregularities in outline. A normal enema, however, does not exclude the presence of intestinal tuberculosis, nor does contraction of the distal colon always indicate the presence of disease. The roentgen method of diagnosis reveals the presence only of intestinal ulceration, but, when associated with pulmonary tuberculosis, especially if the pulmonary disease is at all advanced, it is safe to make a diagnosis of intestinal tuberculosis."

Hammer[9] by means of the barium enema studied the bodies of persons who had died of pulmonary tuberculosis. He removed the distal 20 cm. of the ileum and the proximal 25 cm. of the colon; filled it with contrast material, blew the excess out and inflated the intestine with air; made x-ray pictures of it; and then opened it and compared the preparation with the x-ray picture. He examined 11 bodies. In two cases

there was swelling and infiltration of Peyer's patches with central caseation, but no ulceration; in six cases there was more or less ulceration of the mucosa; in one case there was ulcerative polyposis with productive intestinal tuberculosis; in one case there was ulceration and cicatricial contraction; and in one case there was dysenteriform intestinal tuberculosis. In one of the cases with ulceration there were numerous lenticular ulcers with undermined edges, which did not show in the roentgen picture. Most of the lesions were shown by the contrast fluid; swollen Peyer's patches, which extended over the surface, but were not ulcerated, are, in general, not demonstrable, ulcers are generally recognizable, and when the intestine was inflated with air, marginal ulcers are recognizable in the slightly jagged and wavy contour. Contracted ulcers and scars are generally not recognizable, and lenticular ulcers with undermined edges were not recognizable. Ulcerative polyposis (productive) and dysenteriform tuberculosis appear the same in x-ray pictures as do the pure ulcerative form; the difference is only relative. Changes in the lumen are easily recognizable; and thickenings of the wall are well seen in the x-ray picture. On the basis of these postmortem studies, an attempt was made to demonstrate similar anatomical changes in the living person. For this purpose barium enemas were given when possible and then the bowel was inflated with air. The author studied 34 patients with clinical intestinal tuberculosis. In seven of these cases it was not possible to use any enemas, but in two of these the fine zigzag contour had been demonstrated. In the remaining 27 cases, enemas and inflation with air showed the fine zigzag contour; and six of these cases came to necropsy, and ulcerative intestinal tuberculosis was found in all six.

The laboratory data (other than x-ray) helped but little in the diagnosis of intestinal tuberculosis. Blood and urine examinations proved of no consequence. In our series the stools have been studied for blood, pus, undigested food and amoeba. (Chart I.) Occult blood was frequent. The tendencies to gross hemorrhages from the intestinal tract are few. Fatal cases have been reported. Cruice[10] has collected 26 cases of large hemorrhages and added four fatal cases. Engelsmann[11] reported two fatal cases. Pus occurs only in small quantities. Undigested food is common in the cases suffering from diarrhea. The presence of tubercle bacilli in the stools is of no diagnostic value in view of the fact that in most of these patients the tubercle bacilli can be recovered from the stool because of swallowed sputum.

Pathological studies of intestinal tuberculosis date back to Louis[12] in 1825. Since that time many pathological studies have appeared in the literature. From these pathological reports the incidence of intestinal tuberculosis as a complication in pulmonary tuberculosis varies from about 50% to 85%.

Crawford and Sawyer[13] in a survey of 1,400 autopsies at Fitzsimons General Hospital in Denver, Colo., covering a period of 13 years, report 66.8% tuberculous lesions in the intestinal tract.

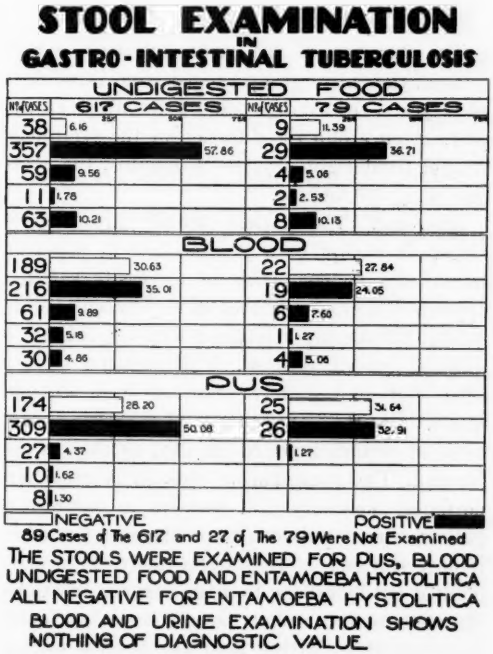

Chart 3.

Goldberg, Sweany, and Brown[14] in 1928, in their pathological studies of enteritis, find ulcerative enteritis in 184 out of 230 autopsies (80%). In their report they also observed 4.3% of this group had peptic ulcer; 20% present submucous gastric hemorrhage; and about an equal number of them showed atrophic gastric glands with atony of the musculature, thinning of the mucosa and flattening of the rugae. In

other words, approximately 44.3% of the 230 autopsies suggested gastritis or peptic ulcer.

In general, tuberculous lesions are commonly found from the mid-ileum to the transverse colon. Tuberculous lesions in the stomach and duodenum are rare, the most common sites being in the lower ileum, cecum and transverse colon. The most common site of the incipient lesions is in one of three places: (a) the lymphoid tissue of the ileum above ileocecal valve; (b) in the cecum at a point where the food current meets the mucosa; (c) the ileal margin of the ileocecal valve.

In our series of 617 cases we have studied in detail during the past six years, 79 of these have come to autopsy. In this paper we wish to compare the diagnostic data in the entire group with the autopsy group.

SYMPTOMS—(CHART 2)

The duration of the gastro-intestinal symptoms in the entire group ranged from three days to 17 years, the average being 14 months. In the postmorten group the duration of the symptoms ranged from eight days to 56 months, the average being 20 months.

Anorexia. The patient usually first notices either an abrupt or a gradual loss of appetite. Oftentimes he complains of an easily satiated appetite which he describes as very "keen" before eating. Frequently associated with anorexia is a sense of gastric fullness. This bloating is very often felt immediately after, or even during, meals. On the other hand, the bloating may recur at certain times of the day, notably in the evening. This symptom we consider of great importance, especially if it happens to occur in a patient who is pulmonally on the mend or even if only stationary. It is quite likely, as already pointed out by others, that the surprise instances of extensive tuberculous bowel ulceration found on postmortem, having been unsuspected during life, would be fewer if more attention were paid to the above described anorexia with the frequent concomitant bloating and resort be had to a more thorough history and physical examination.

Vomiting. Vomiting, if present, is seldom of the distressing type. Its interpretation is, however, at times difficult, since it may appear as an emetic cough; if unaccompanied or uninitiated by cough, even if not frequent, it should arouse suspicion. Repeated copious vomiting immediately or some time after meals and persisting over a long period is probably not of intestinal origin. Thus in one of our cases, it was associated with pain in the epigastrium and ileocecal region. The vomiting persisted with varying degrees of severity for a period of six months. The patient was put on tuberculous enteritis management, which rapidly proved quite effective. Two years later the patient died during a frank pulmonary hemorrhage. On postmortem perihepatitis and perisplenitis were found, but no intestinal involvement. Spells of intermittent vomiting with or without nausea should always be looked upon as of serious prognostic significance. Nausea with or without vomiting may persist at an irregular time of the day, most frequently in the morning.

Pain. Pain as a symptom in specific enterocolitis is of great significance for it is most constant. It is seldom very severe and may occur early in the disease. In fact, it is very often only a tenderness elicited on palpation or motion rather than an actual pain. The location of the pain while frequently found to be in the ileocecal region, in some cases it quite often is—at least initially throughout the entire course of the colon, the midepigastric region or the lower left quadrant of the abdomen. It is obvious that even in the unusual instances the ileocecal region, the classic site of tuberculous involvement, may eventually become the seat of pain. Frequent cramp-like, general abdominal pain may occur, especially during a diarrhea siege, but seldom is it continuous or even prolonged.

If at the onset the midepigastrium chances to be the location of the distress, it occasionally simulates peptic ulcer or cholecystitis. However, bearing in mind the time relation of the pain, the food intake, the rhythmic occurrence of remissions, the usual absence of associated anorexia, the character of the pain being more than mere tenderness—while in tuberculous enterocolitic the pain may occur during eating or immediately after eating—again it may be relieved by bowel movement, and at times it is severe either before or after a bowel movement. Then the greater frequency of intestinal tuberculosis in the pulmonally tuberculous should be borne in mind. On the other hand, the oversight of a peptic ulcer in the tuberculous is not to be condoned. Hence repeated x-ray examinations should be had in doubtful cases.

Cholecystitis is another prolific source of diag-

nostic confusion when the pain is epigastrically located. As a matter of fact, it may even coexist. However, in the average early case the absence of a history of periodic attacks, the absence of rigidity and pain in the right hypochondrium, a negative Graham test suffice to differentiate the two conditions. Parenthetically it may be added that in the late cases the rigidity of the ileocecal region usually propagates itself to the right hypochondrium. But in these cases the existence of intestinal tuberculosis is not questioned.

The ileocecal site of pain is not devoid of diagnostic difficulties, especially when the onset is acute. In that case it is impossible to differentiate from ordinary acute appendicitis, since it invariably presents the initial generalized and the subsequent localized abdominal pain syndrome. Moreover, it almost always presents a high leucocytosis and an augmented polymorphoneucleosis. The fever curve is nearly always identical with that met with in the nonspecific appendicitis attack.

Except in exacerbations of well established intestinal tuberculosis we always advise operation. At one time Smithies, et al[15] cherished the hope that in case it should prove cecal tuberculosis, extirpation of the area carrying the brunt of the disease, allowing of course for a solitary tubercle here and there to remain behind, would eventually result in a cure. They found in early subacute or chronic cases where operation was done that the physical findings were no criterion of the actual extent of the involvement. Not only the cecum but also the ascending colon and even the terminal ileum were found studded with nodules so as to require an altogether too formidable bowel resection.

Summary of Pain. In general, the pain may be acute, stabbing, boring, or cramp-like in character, or again may be only a slight soreness or dull aching. The pain is most frequently localized in the right and left lower quadrant of the abdomen or somewhere along the course of the large bowel. Again it may be diffused over the entire abdomen or localized to the epigastric region or entire upper abdomen. The pain usually is made worse by eating and is usually relieved following a bowel movement or vomiting. Cold drinks and cold foods may initiate a pain or increase the intensity thereof.

Diarrhea. Diarrhea may be a very early or a very late symptom; may be persistent, intermittent or alternating with constipation; may be painful one time or painless at another time. Its outstanding features are that it is always exhausting as it occurs and emaciating in the course of time if not counteracted by treatment. An untreated, frequent or prolonged diarrhea that does not prove exhausting and emaciating is, most likely, of nonspecific origin. The frequency of the stools may vary from one to twenty. The average case suffering from diarrhea would complain of from five to six bowel movements daily during the attack of diarrhea. These attacks may last from one day to several weeks. The patient suffering from diarrhea rarely complains of copious pus or blood in the stools. A sense of incomplete evacuation is commonly complained of during the period of loose bowel movements and may prove, at times, very troublesome.

Intestinal Hemorrhage. Intestinal hemorrhage as a result of intestinal tuberculosis is rare in our series. In the small percentage of cases where gross bleeding occurred, it was found to be due to focal rectal conditions such as hemorrhoids, fissures and thrombosed veins, carcinoma or nontuberculous ulcerative colitis. A history of gross bleeding interpreted as being due to intestinal tuberculosis occurred in only two cases in our series.

Constipation. Constipation varied from a mild form, relieved by small amounts of mineral oil, to a severe constipation without bowel movement for two or three days. Attacks of severe constipation may be followed by spells of diarrhea or again may persist throughout the entire course of the disease.

Gastric Symptoms. In a careful study of the pathological reports, one is inclined to believe that various types of gastritis are present more frequently than is clinically elicited. An accurate knowledge of the gastric involvement can only be obtained by the use of the gastroscope, and this we hope to add to our diagnostic equipment in the near future. The many gastric symptoms, such as bloating, fullness, belching, sour stomach, heart burn, acid eructation, distress simulating peptic ulcer, slight gastric hemorrhages, in the absence of x-ray findings, all point to a frequent occurrence of a probable gastritis. An early study of the gastric mucosa by means of the gastroscope in the patient suffering from a pulmonary tuberculosis may glean some very interesting facts.

PHYSICAL FINDINGS—(CHART 3)

With few exceptions the cases under observation were all suffering from far advanced pulmonary tuberculosis. The average age of the entire group of the males was 31 years and the females 27 years and six months. In the postmortem group the average age of the males was 27 years and nine months, and the females 24 years and nine months.

Loss of weight or failure to gain under proper treatment occurring in a patient in whom we can discover no extension of the original pulmonary lesion, may herald the onset of intestinal tuberculosis, provided, of course, we ruled out the various conditions such as focal infections, hyperthyroidism, diabetes, malignancy, an activated latent heart lesion or tuberculous kidney. In our entire series, 408 give a history of weight loss (66.13%), ranging from one to 80 lbs., the average weight loss being ten lbs.; 93 gained weight (13.07%), ranging from ¾ to 41 lbs., the average being 9¼ lbs.; 32 remained stationary (5.18%); 84 (13.62%) were incomplete. In the postmorten group 48 (60.76%) showed a weight loss ranging from three lbs. to 50 lbs., an average of 18¾ lbs.; seven (8.86%) gained weight, ranging from one to 11 lbs., an average of 3½ lbs.; two (2.53%) remained stationary; and 22 (27.85%) no record, weights probably not taken because of the condition of the patient.

Pallor is also very suggestive. It does not, as a rule, mean a true anemia. Pulmonary tuberculosis alone seldom presents that yellow grayish shallowiness met with in the untreated, advanced intestinal tuberculosis.

Ascites we found rather uncommon in our cases. When it did occur, the presence of intestinal involvement had already been established. On the other hand, the origin of the ascites itself was sometimes in doubt. For our purpose we have made it a rule to regard in the intestinally tuberculous a slow oncoming, moderate ascites showing a tendency to resorption under treatment as of tuberculous origin; a rapidly forming, copious ascites requiring frequent tapping with no tendency to resorption is either entirely of non-specific origin—i. e., cardiac, renal, or hepatic, conjoint as a superimposed factor.

Of the physical signs the most useful is that of rigidity in the ileocecal region. It is usually not of the board-like type. The right lower quadrant and very often the entire right rectus presents a "leathery" feel or a sheer bogginess. The ileocecal region reveals a fullness that renders it asymmetrical with the gentle slope in the contralateral region. On palpation, one frequently meets with an ill-defined, irregular, yielding, moderately tender and at times ballooned mass.

The scaphoid abdomen is met with usually in the very late cases where the diagnosis is seldom in doubt. In such cases we found the localized rigidity nearly always absent. The abdominal muscles have a "granular" feel. The skin appears "stretched" and feels like tissue paper.

The anal sphincter is very often unusually rigid, frequently because of associated fissure, fistula or ulceration immediately above or in the vicinity.

Aiming to distinguish between tuberculous diarrheas and those of amebic dysentery and ulcerative colitis, we, in every case, on the initial examination order feces and proctoscopic examinations. The former establishes the presence or absence of amebiasis. The mere prescence of tubercle bacilli in the feces is not conclusive since it may come from swallowed sputum.

Proctoscopic Examinations. In cases in which low colon ulceration is due to tuberculosis, the usual proctoscopic appearance is fairly characteristic. The ulcers are moderately large; they vary from a few millimeters to several centimeters. Their outline is irregular and the edge is usually reddened, thickened and slightly raised and often undermined a distance of a few millimeters; the undercutting may be extensive. Commonly, the ulcer base is covered with a yellow pyogenic membrane which is only slightly adherent and on its being swabbed away, tuberculous granulation tissue is revealed. The granulations bleed easily but, when dry, appear finely nodular or pebbled. Typical lesions can usually be diagnosed tuberculous from their proctoscopic appearance alone; the atypical forms present difficulty.

Not all tuberculous ulcers exhibit all the characteristics just mentioned; healing ulcers, their bases covered with a clean gray membrane, may be interspersed with the active lesions in all stages of progress. The mucosa between the ulcers may be edematous, often reddened and at times dry and atrophic. It is generally normal except for vascular changes. Moderate diffuse redness is common; in some instances areas paler

than normal are interspersed with areas redder than normal. This phenomenon gives a diffusely mottled appearance to the bowel wall. The engorged mucosa itself does not bleed when it is swabbed with a cotton-tipped applicator but the fine venules in it may, with a resultant line of oozing marking the vessel's course. In arrangement these vessels form branching (tree-like) or parallelogram (diamond-shaped) patterns in the mucosa. While at times such may be visible with any type of acute or subacute colitis, they are fairly constant in tuberculous colitis. They are not visible in all cases.

In the tuberculous lesions, loss of mobility of the portion of bowel visualized by the proctoscope is not common. Mucosal ring stricture may occur at the site of a healed or healing ulcer but tubular stricture is rare. Diffuse contraction of the ampulla and diminution in the size of the rectal valves is rarely seen, unlike the process in chronic ulcerative colitis.

The roentgen studies were done almost exclusively by means of barium enema. The enema used was a mixture of barium-kaolin. This mixture was resorted to in order to minimize the bowel irritation. In many instances after the first bowel evacuation following the administration of the barium mixture, the bowel was inflated with air. With few exceptions, using this method, the patients suffering from tuberculosis rarely complained of discomfort while the colon is filled and do not object to the periods of delay between observations. Mass movement is very seldom seen even in the well advanced tuberculous enterocolitis.

The zigzag, or "mouse nibble," contour denoting tuberculous ulcers, as described by Hammer,[16] were also observed by us, as was also the irritability of the bowel and the failure of the cecum and other proximal parts of the colon to fill and retain barium. Many of the patients observed had residue-filled loops, probably as a result of an established cathartic habit. It is our experience that only too often a bowel which is considered normal, as visualized by the barium enema method, does not exclude the presence of tuberculous enterocolitis.

No serious attempt was made in our study to separate our cases into enterogenous and hemotogenous groups. The fact is that these groups are not sharply defined. The same case may at one time or another belong to either or both. In the late generalization of the chronic localized case the intestinal involvement may assume an hemotogenous aspect akin to the lesions in the other organs sharing in the widespread invasion.

The temperature curve, if high and continuous and if not accountable by either an increase in the pulmonary or distant-organ involvement, may conceivably point to a hemotogenous origin in an existing intestinal involvement. However, postmortem material does not confirm this view.

Rather than the above alluded to systemic reaction, one is inclined to regard the local manifestations as the more decisive. Thus, given a diffuse, non-localizing pain over the entire colon accompanied by the usual other symptoms, i. e., diarrhea, loss of weight, etc., plus a rapid course, one would be inclined to regard the process of hemotogenous origin. Even then, it is, perhaps, highly questionable.

Of the entire series, representing 617 cases that were considered positive for intestinal tuberculosis either by x-ray or clinical observation, or as found at postmortem, 543 that were considered clinically positive were diagnosed positive by x-ray in 404 instances (74.4%); negative in 139 instances (25.6%). The proctoscopic examination was positive in 29 of 481 cases (6.02%).

Of the 79 postmortem cases, only 60 had a complete examination, including x-ray diagnosis. Of the 60, 50 had tuberculous involvement of some portion of the gastro-intestinal tract (83.33%). The clinical diagnosis was correct in 48 cases (80%). The x-ray was correct in 38 (63.33%). The proctoscopic examination was positive in three cases (5%). In the 79 postmortem cases the clinical diagnosis was correct in 62 instances (78.6%). A careful analysis of the 17 cases that were considered incorrect revealed that nine had pronounced tuberculous changes in the liver and spleen. The symptoms in this group closely paralleled those in which ulcerative lesions of the intestinal tract were found. The tendency to more prolonged vomiting seemed evident in this group — perhaps a point of differentiation.

SUMMARY

(1) There is no specific procedure as yet available in the accurate diagnosis of intestinal tuberculosis. The clinical observations are still the most dependable method for the detection of intestinal tuberculosis. In our series the clinical diagnosis proved correct in 80%.

(2) The roentgen studies by the barium enema method proved of value in 63.33%.

(3) The protoscopic examination was a direct aid in 5% of the cases that came to postmortem and 6.02% in the entire series of 617 cases.

(4) The stool examinations helped to exclude nontuberculous enteritis and amoebiasis. The examinations of the blood and urine were of no help.

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BIBLIOGRAPHY

1. Brown; Lawrason, and Sampson, H. L.: Intestinal Tuberculosis, 154-204, Phila., Lea & Febiger, 1930.

2. Engelsmann, R.: Ueber die sekundäre Darmtuberkulose, Beitr. z. klin. d. Tuberk. **38**: 16, 1917.

3, 4, 5. Lemon, W. S.: The Status of Present-day Methods of Examination in the Diagnosis of Intestinal Tuberculosis, Minnesota Med., **6**: 300, 1923. Tuberculosis Enterocolitis, Minnesota Med., **6**: 572, 1923. Intestinal Tuberculosis: III. Clinical Studies Relating to Diagnosis, Trans. Nat. Tuberc. Assn. **21**: 186, 1925.

6. Logan, A. H.: Chronic Ulcerative Colitis; a Review of One Hundred and Seventeen Cases, Northwest Med. **18**: 1, 1919.

7. Stierlin, Eduard Die Radiographie in der Diagnostik der Ileozoekaltuberkulose und anderer Krankheiten des Dickdarms, Munchen. med. Wchnschr. **58**: 1231, 1911.

8. Brown; Lawrason, and Sampson, H. L.: Diagnosis and Treatment of Tuberculosis of Small and Large Intestine, J. A. M. A. **98**: 26, 1932.

9. Hammer, G.: Die Roentgendiagnose der Darmtuberkulose mit Untersuchungen uber die Funktion Valvula Bauhini, Deutsch. med. Wchnschr. **54**: 57, 1928.

10. Cruice, J. M.: Intestinal Hemorrhage in the Tuberculosis, Med. Rec. **84**: 471, 1913.

11. Engelsmann, R.: Ueber die sekundäre Darmtuberkulose, Beitr. z. klin. Tuberk. **38**: 16, 1917.

12. Louis, P. C. A.: Recherches anatomico-pathologiques sur la phthisie, Paris, Gabon, 1825; Recherches anatomiques, pathologiques et therapeutiques sur la phthisie, 2. ed., Paris, Bailliere, 1843.

13. Crawford, Paul M., and Sawyer, Harold P.: Intestinal Tuberculosis in 1400 Autopsies, Am. Rev. Tuberc. **30**: 568, 1934.

14. Goldberg, B.; Sweany, H. A., and Brown, R. W.: Pathological Studies on Tuberculosis Enteritis, Am. Rev. Tuberc. **18**: 744, 1928.

15. Smithies, Frank: Weisman, Morris, and Fremmel, Frank: Tuberculous Enterocolitis, J. A. M. A. **91**: 1952, 1928.

16. See 9.

DISCUSSION

Henry C. Sweany (Chicago): Mr. Chairman, Members of the Section: It may seem rather odd that a laboratory man should open the discussion on a paper about which he has absolutely nothing to contribute toward a diagnosis. It is true that the bacteriological examination may reveal acid-fast bacilli, but anybody may show acid-fast bacilli in the stool in connection with positive sputum; so it contributes nothing. It is true that examination for blood will assist in showing if it is an open lesion in the intestinal tract, but that is a common condition. So we come to you with empty hands so far as diagnosis is concerned. But in the anatomical studies the pathologist has something to contribute.

From the standpoint of anatomy we have constrictions along the gastro-intestinal tract that lead to a basis of diagnosing of the various symptoms. At the pylorus there is a constriction, but there is too much acidity for the tubercle bacilli to develop. In the bowel there is also a constriction at the ileocecal valve, but the concentration of H-ions is such that the tubercle bacilli are capable of penetrating the lymphatics and setting up growth.

In the isolated follicles these bacilli penetrate either by direct extension or else by being pushed in there during the constriction at the ileocecal valve.

The next point of stricture is the rectum, where there is an accumulation of material and the bacilli are quite frequently forced into the lymphatics above this point. There are, therefore, various anatomical features which lay a foundation for the development of the lesions. Remember that tuberculosis of the bowel is like that of the lung, primarily a lymphatic disease. It enters the lymphatics, sets up the early ulceration, and the first thing found is a longitudinal ulcer in the region of the ileocecal valve. Rarely do you see the primary ulcer in any other position of the gastro-intestinal tract. The ulcers gradually extend in each direction from the ileocecal valve, depending on the severity of the condition. As it becomes worse they go toward the pepunum above and toward the transverse colon below, nearly always beginning in the region of the ileocecal valve. Later on these ulcers in the Peyer's patches spread around the bowel and produce the advanced annular ulcers.

Sometimes the lesions are caused by direct trauma, due to an injury produced on the wall of the cecum or ileocecal valve, by direct contact of fecal material passing through.

I think the greatest of all the diagnostic criteria, from a clinical standpoint, is pathological-physiology. It is true these ulcers form a diagnostic basis for x-ray examination, outlining the ragged type of colon and ilium, but the pain and all the other signs that lead to diagnosis are due to a pathologic physiology.

Here we have a great deal more difficulty in analyzing the conditions. We all know the two nerve plexus, the Meissner plexus of the submucosa, and the Auerbach plexus of the muscle layer—the principal nerve distributions of the ilium and colon.

If there is a stimulation of the vagus there is more liable to be a diarrheal condition, and a stimulation of the sympathetic is perhaps more liable to produce a temporary constipation. These two may alternate. You may have a segmental irritation in a small portion of the bowel, where paralysis with flatus and other symptoms may occur. Deep and superficial pain may result. All these things play a very important role in the diagnosis.

Finally, you may have the absorption of toxin from this ulcerated bowel which produces various effects, some of which are loss of appetite, rise in temperature, etc. That, of course, produces loss of weight. Stimulation here again of the sympathetic nervous system will produce a dilatation of the vessels of the whole mesentery, and there results so-called "internal bleeding within the blood vessels" which draws blood to the

mesentery and causes a pallor on the superficial skin surface.

This subject of gastro-intestinal tuberculosis offers one of the most complex problems for accurate diagnosis of any subject there is in medicine, and brings into play practically every aspect of pathology and pathologic physiology in the clinical diagnosis so well given by the authors.

Dr. Morris Weissman, Chicago: We did not attempt to diagnose tabes mesentericus because we expected no corrobation on postmortem. At post, tabes mesentericus alone is seldom encountered. In its late stages the intestines are so overwhelmingly involved as to dominate the picture. The glandular component is at best represented only as the "burned ashes" of an initial process. However, in general it may be stated, given a case with vague nonlocalized abdominal distress with frequent febrile exacerbations and remissions over a prolonged period of time, especially so if associated with a predominantly hilar and pleuritic type of pulmonary tuberculosis-tabes, mesentericus may well be suspected. The diarrhea attacks that generally occur may be construed as reflex. Supervening terminal meningitis in a case of intestinal tuberculosis may well suggest the coexistence of tabes mesentericus for the interrelation of glandular processes and meningeal involvement is well known.

Certain acute gastro-intestinal conditions may temporarily prove confining. Phrenicotomy, for example, does at times initiate a train of gastro-intestinal symptoms that may prove baffling. Acute pleurisy with effusion, to cite another example may, in the course of its development present severe gastro-intestinal distress. But, bearing in mind their acute episodal nature plus the fact that vomiting is an outstanding symptom readily differentiates them from the more permanent and relatively less acute intestinal tuberculosis in which vomiting is of lesser prominence.

A word about certain tuberculous types with reference to their bearing on extrapulmonary manifestations. These types by their appearance give clue to the organs involved. Thus the flushed—the vasodilator type—frequently shows pulmonary involvement alone. While the process is frequently attended by great toxicity, it usually eventuates in fibrosis. Extrapulmonary tuberculosis is less common and slow in developing if it does occur. The pallid—the vasoconstrictor type—very often spells extrapulmonary involvement, intestinal tuberculosis being the most common. The process is apt to be widespread and with little tendency to fibrosis. Finally the blue—the asthmatoid-passive congestion type—where the extrapulmonary organs affected are the heart, primarjly as myocarditis and the liver and kidneys secondarily. Dr. Hruby, Secretary of the Board of our Sanitarium, aptly sloganed these clinical pictures Red, White and Blue. He suggested that we undertake at some future time to tabulate and correlate the types with the multivaried clinical manifestations met with in our patients. As yet we have no statistical data to substantiate the aforestated impressions.

PLASTIC AND RECONSTRUCTIVE SURGERY ABOUT THE FACE AND HEAD—THEN AND NOW

JOSEPH C. BECK, M. D.

CHICAGO

It was in the early years of my practice that I began this work in plastic and reconstructive surgery. My first paper was presented in Pittsburgh at the Carnegie Institute before the Triological Society and the question was raised by the Council as to whether or not they would permit me to present it, because there was a doubt in their minds as to whether this phase of surgery belonged to the activities of the "quack." Such cases of esthetic surgery as I showed were not being performed by regular medical men. I was very grateful to Dr. Richards of Fall River, Mass., a member of the Council, through whose efforts I was permitted to read my paper entitled, "Plastic and Reconstructive Surgery About the Face." From that time on there was much of "Love's Labor Lost" in this type of work. The general men and some of the earlier surgeons, especially in Europe, did do some of these operations. The Italian operation, such as binding the arm to the face, was one of the earlier more popular procedures, but the more direct work about the face and ears was performed at a much later period. It was not until twenty years ago, about the time of the World War, that esthetic plastic work came into its own. Today we may be quite proud of the excellence of this work and the splendid results obtained. I also desire to mention the formation of a Board of Examiners who pass upon the ability of the men in this field.

The most interesting part of the work has been the refinements that have been made in rehabilitating individuals who have suffered terrific trauma particularly following automobile accidents. We always insisted upon obtaining a definite history because these people were timid about taking risks as to the final cosmetic results. One was frequently threatened with lawsuits and was accused of having done something that was not acceptable to the patient. In the beginning it was our custom to take stereoscopic pictures of every patient throughout the course

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and steps of the operation. Not only pictures, but casts of the face were made so one had something to go by in comparing the results with the initial findings before operation. Some people we actually declined to operate on because one could see that these patients were neurotics

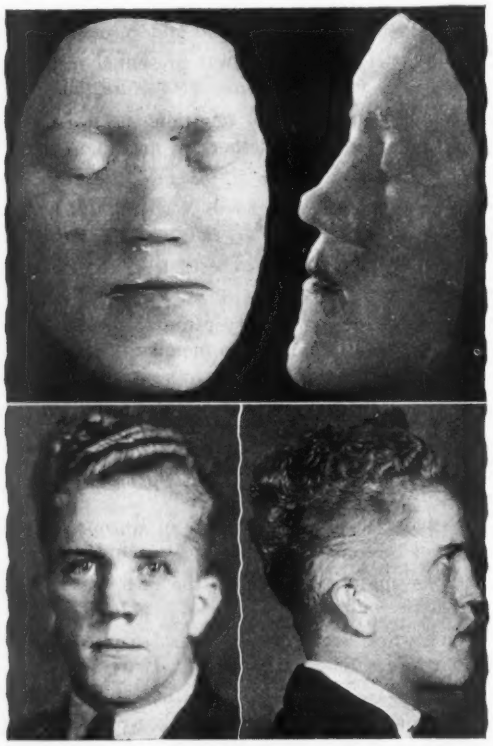

Figure 1. Repair of poorly reduced nasal fracture. Patient was so sensitive about his appearance that he would not permit preoperative photography.

and would never be satisfied no matter how perfect the procedure would be. One important element in the early period was the question of syphilis. We did not have the Wassermann test then and we did operate on a large number of syphilitic patients, some of them having most terrifying lesions. It was surprising how comparatively little difficulty there was encountered in the healing process of these plastic cases.

The old time techniques of plastic and reconstructive surgery can now be considered a thing of the past and the newer methods that have taken their place, especially in luetic cases, is well worth while emphasizing. While we had many disappointments in some plastic cases, the end results obtained then, compare very well with that which one gets now.

I seldom saw a patient who had a great deal of deformity requiring much work who was not grateful for what was done for him, even though the esthetic result was not altogether perfect. Those cases that had minor defects were troublesome to us and still are. There was often a psychic element in that type of patient as I have already stated above.

At this point I should like to say a word about Military Plastic Surgery. During the last war we were unprepared in reconstructive and plastic surgery but today, the United States Army Medical Corps has sufficiently trained plastic surgeons, who are able to give meritorious service in this special branch of miltary surgery.

Now my associate, Dr. A. M. Berman, will present that part of my paper dealing with the more modern thought of reconstructive surgery, perhaps repeating some of the things I have said in my preliminary remarks, but bringing up the subject to the present day acceptance by the profession. In my closing remarks I shall have something to add regarding plastic rubber protheses.

PLASTIC AND RECONSTRUCTIVE SURGERY OF THE FACE AND HEAD NOW

The slow but steady progress of plastic and reconstructive surgery has been watched with keen interest by medical science the world over. From a humble beginning in barber shops, hotel rooms and beauty parlors, plastic surgery has

Figure 2. Saddle Nose repaired with cartilagenous implant.

now come to be a recognized specialty, practiced by men who are thoroughly grounded in the principles of present day aseptic surgery. With the ever increasing industrial hazards and traffic accidents, has come an increasing demand for more

efficient reconstruction of maimed parts especially about the head and neck. American surgeons, especially since the World War, have taken a great interest in plastic and reconstructive surgery, and have kept pace with and in many instances even surpassed the skill and artistry of their European colleagues. The quacks and charlatans who have practiced so-called "Cosmetic Surgery" in the past, are rapidly disappearing from the horizon, and their places are being taken by thoroughly trained surgeons who are at last really practicing reconstructive surgery as it should be.

In discussing plastic and reconstructive surgery about the head, it might be well to state that reconstruction of the nose is the most common operation we are called upon to perform. Just as in other phases of surgery, the techniques are fairly well standardized at the present time. The sawing off of a hump on the dorsum, or the elevation of a depressed tip is with very few exceptions a standard procedure and can be found in any recognized text. One of the important phases of rhinoplasty is the repair of a nasal fracture which is so commonly met with today. Straith, DeKleine, Safian and others state that nasal fractures must receive immediate attention and careful reposition of all parts of the nose must be accomplished as soon after injury as possible. Adequate metal splinting devices or mattress sutures passed through the skin and secured by buttons may be used to maintain the position of the parts. If there has been a loss of tissue

Figure 3. Alar loss repaired by flap from forehead.

from the ala or dorsum of the nose, sliding or tubular grafts may be employed. Care must be taken, however, to accurately match the skin texture and color to the surrounding area. There has been some diversion of opinion as to whether it is best to repair loss of skin by grafting immediately after injury, or to wait until some later date to effect a repair. Straith believes that loss of tissue should be immediately replaced by a Wolfe Graft. One should remember also that in the repair of a nasal fracture, one should not only take care to effect a pleasing esthetic result, but

Figure 4. Repair of hare lip.

also to obtain a proper airway in the nose. No rhinoplastic procedure is complete without thorough investigation and repair of component parts of the internal nose. (Fig. 1)

Depressions of the dorsum of the nose has offered many problems to the plastic surgeon in the past. At the present time, we treat this deformity by employing cartilage grafts. We use either a piece of rib implant from the patient or some cartilage from a donor. In many institutions cartilage implants from other persons are kept in an aqueous solution of merthiolate in a refrigerator, and these are ready for instant use. Many of us still use ivory implants and although the use of foreign substances in tissues has been decried by many, ivory implants are still used with good results. More recently there has been reported the use of temporary ivory implants in the nose, which is later supplanted by cartilage. It is said that the ivory when left in situ for about six months to a year tends to make a better bed for the reception of the cartilage implant later. The use of bone implants has been discontinued by some surgeons because it was felt that bone had a tendency to atrophy. Although bone is still being used by many, we feel that cartilage is far superior. (Fig. 2).

When there is a loss of an ala, sliding or tube grafts are used to repair the defect. (Fig. 3). The cosmetic end results of such a procedure is not always satisfactory. Recently a new type of rubber prothesis was developed which is far superior in many cases to surgical repair. In cases

where the ala has been torn away, immediate and accurate suturing of the parts gives most excellent results.

Many patients, men as well as women, frequently consult the plastic surgeon regarding a plastic operation in order to satisfy their vanity

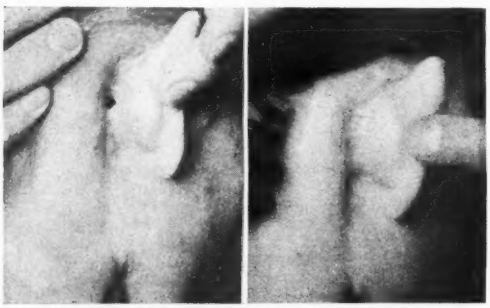

Figure 5. Repair of post-auricular fistula.

although the deformity that exists is very slight. In the past we have turned these people away because we neglected to take into consideration the fact that even slight deviations from the so-called normal may have a decided effect upon the individual. In recent years, plastic surgeons in conjunction with psychiatrists have studied the psychological consequences of disfigurement on individuals and have come to the conclusion that regardless of how trivial a deformity there may be, that deformity should be corrected if the patient considers himself handicapped either socially or economically. It has been shown that disfigurement about the face is a serious handicap in securing employment, especially in those jobs which necessitate meeting the public. Frequently we find that children with average or better than average mentality are failures in school because they possess some form of facial deformity which makes them the object of ridicule among their classmates. It is gratifying, however, to see these individuals rehabilitated and returned to usefulness through plastic surgery without which they would have been doomed to a life of failure and disappointment.

In the correction of a deformed nose the mastery of the technique of the operation is not the only consideration. The plastic surgeon, if he is going to succeed in pleasing all of his patients, must also have artistic ability and a delicate sense of proportion. A small pert nose on a heavy jawed, large face does not produce a pleasing esthetic effect. Formerly little attention was paid to molding the nose to suit the individual personality and as a result the plastic surgeon was plagued with lawsuits. Now we study each case by photographs, casts and actual meticulous measurements with specially devised instruments and determine before operation just what type nose would suit that particular individual. It is here that plastic surgery becomes, what Maliniac has so aptly called "Sculpture in the Living." There are many instruments devised for measuring relations of the nose to the rest of the face. Essentially these instruments are variations of several types of protractors. As a general rule the profile line should be at an angle of 25 to 35 degrees, and the length of the nose should never exceed a line drawn from the root of the columnella to the tip of the chin. In women, especially, it is better that the length of the nose should be shorter than the chin line. Another important measurement in determining a pleasing esthetic appearance is the determination of the angle between the upper lip and the columnella. Safian calls this the septo-labial angle and has shown that it should be between 90 and 105 degrees to be normal. After all measurements are accurately determined and marked off on the patient's photographs and casts a careful psychiactric appraisal of the patient is begun. A thorough physical examination together with complete blood analysis should be

Figure 6. Lip deformity due to lymphangioma. Child refused to attend school until the deformity was corrected.

completed before the patient is admitted to the operating room. Careful surgery with exacting care given to each step in the operative procedure insures a successful end result. The postoperative care in these cases is most important because if this phase of the procedure is neglected

a good operation can be ruined by infection or inadequate splinting devices.

Another important phase of reconstructive surgery is the repair of hare lip and cleft palate. We like to operate on these cases as soon after birth as possible. By experience we have learned that the best results are obtained when early surgery is performed. (Fig. 4). Hare lips have been repaired with most excellent results as early as twenty-four hours after birth. Although the repair of hare lips and cleft palates falls into the realm of plastic surgery, the oral surgeons are taking over more and more this type of work, and rightly so, because of their superior knowledge of the mechanics of the oral cavity.

In accidents where there has occured lacerations about the face immediate plastic repair of the wound should be done. Haphazard suturing results in scar formations and necessitates a secondary operation for their removal. Every facial laceration should and must receive meticulous approximation of skin edges and adequate postoperative splinting if scars are to be avoided. In those patients in whom we feel there is a tendency to keloid formation superficial x-ray irradiation to the suture line may prevent thickened scars.

The plastic surgeon is frequently called upon to correct abnormally protruding ears. This is a simple procedure which offers no difficulty to the skilled operator. If a crescent shaped piece of cartilage is excised and a new antihelix is formed the spring action of the auricular cartilage is removed and the ears are thus permanently held back. Strict attention to asepsis and snug postoperative dressings preclude the possibility of chondritis and hematoma in these cases.

Rudimentary ears (microtia) and atresia of the ears are still a great problem to the plastic surgeon. Although many cases of reconstructed ear deformities have been reported as excellent results actually they are far from being good. For the most part congenital deformities of the ear lend themselves poorly to plastic reconstruction. Here, again, at least for the present, plastic rubber protheses correctly made, offer the best esthetic results.

Post-auricular fistulae, as a result of continuous and prolonged packing of the mastoid wound following mastoid surgery is not so commonly met with these days. Now and then a case of this type presents itself for repair. These fistulae are repaired by making a post-auricular incision and splitting the flap in two layers. The inner layer obliterates the mastoid cavity while the outer layer closes the external defect. (Fig 5).

The correction of deformities resulting from tumors both benign and malignant, offers many problems to those practicing this specialty. Space prohibits a technical discussion of deformities resulting from these new growths but suffice it to say that these cases offer most interesting material and ofttimes most excellent plastic results. Cavernous hemangiomas and lymphangiomas although causing marked deformity are readily amenable to correction. Fig. 6 illustrates a case of lymphangioma of the upper lip which was operated upon with most gratifying results.

Figure 7. Large facial defect due to radical operation for carcinoma of the antrum and showing restoration by rubber prothesis.

When defects of the face are large, such as a result of radical operation for malignant diseases of the antrum of Highmore, we use rubber protheses to best advantage in restoring the normal appearance of the patient (Fig. 7). Experience has shown that these cases cannot be restored properly and satisfactorily even with multiple plastic procedures.

Facial paralysis is a deformity which tests the acumen of every plastic surgeon who attempts a correction. Our latest method of correcting this deformity is that of bridging across the defect in the facial nerve with a nerve graft from one of the superficial femoral nerves. Although end to end nerve anastomosis is still practiced the nerve graft method gives better results. Bell's palsy is relieved by removing the bony covering of the nerve canal which prevents pressure atrophy due to edema of the nerve. The decompression operations should be done early if per-

manent paralysis is to be avoided in these cases.

In the practice of reconstructive and plastic surgery one is frequently obliged to correct a receding chin or a protruding jaw of prognathism. Many cases of chin recession can be corrected by introducing ivory, celluloid, or cartilage subcutaneously. Here again cartilage is to be preferred to foreign material implants. In more complicated cases of receding chin, operations upon the mandible and displacement of the condyle may have to be done in order to reconstruct the jaw properly. Great care and caution must be exercised in these cases because not only should one try to achieve a good esthetic result but the biting surfaces must also be carefully preserved. In reconstructing the receding chin or the prognathic jaw, the well trained oral surgeon has a better advantage over the plastic surgeon in many cases, especially where the mechanics of chewing must be restored to normal.

In conclusion we wish to state that plastic and reconstructive surgery offers vast relief from mental anguish and psychic distress and in many cases rehabilitates individuals to a normal useful, social and economic life who would otherwise consider themselves misfits of society because of facial deformity which are either congenital or acquired.

Closing remarks by Dr. Joseph C. Beck: I desire to show some of the newer type casts and moulages and some new material used in making protheses which has been developed by Mr. Gardner of Chicago. This work is being done for us by Mrs. Hall in our office This (demonstrating) is the latest possible substitute we can offer to patients with complete loss of the external ear. It is much better to have these protheses because one may operate as often as fifteen times, as I did in one case, and still have nothing but a deformity at the end. The protheses are very important in connection with plastic surgery about the face, especially following complete loss of an eye and eyelids, the external nose or any part of it, the whole of the superior maxilla as following resection operations for cancer, or extreme destruction from trauma and infection. (Demonstrating a model of same).

Plastic surgery is now on a plane of such refinement that a man who does a great deal of it will obtain results that at times are unbelievable. I only wish that I was beginning my work in plastic surgery. However, I take great pleasure now in indicating operations, directing and outlining the procedures in my practice to my associates who received their training from me, and seeing the work done properly.

TRAUMATIC PSYCHOSES

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From the Chicago State Hospital, Edward F. Dombrowski, M. D., Managing Officer

CHICAGO

With the increasing frequency of automobile accidents, the speeding up of traffic, increased mechanization of factory work and with a new era in alcohol consumption, the question of head trauma has become correspondingly important. There is no other part of the body which, following trauma, is as likely to be followed by death or disability of a severe nature.

In an institution for mental patients, we are concerned with that aspect of head trauma which deals with the mental and neurological manifestations of head injuries, but particularly those mental aberrations which made institutionalization necessary. However, the problem of traumatic injuries of the head is not limited to its medical aspects, for in addition to the medical problem of immediate injury and its sequelae, there are the problems of litigation, which so commonly follows these injuries; the conflicting nature of medical opinions in the courts, human suggestibility, and finally the malingering of some persons with intent to defraud.

Although primarily this discussion will concern itself with the psychoses following head injury, it is believed that a discussion of this kind would be incomplete without first mentioning the so-called traumatic neuroses. Wechsler described these neuroses as cases in which there was no early or late evidence of brain injury, but which were followed by subjective complaints with few organic signs at the time of the accident or afterwards and no evidence of brain injury later. Four subgroups were listed by Wechsler. All of these were associated with subjective symptoms such as headache, vertigo, memory defects, fears, anxieties, vasometor disturbances, tremors and inability to work.

Read before Physician's Association of Department of Public Welfare at Illinois State Medical Society, May 2, 1939, Rockford.

Obviously, patients with traumatic neuroses are not committed to a state institution unless the symptomatology becomes so pronounced that there is a question of a borderline psychosis. This study was undertaken with the viewpoint of classifying some of the factors pertinent to the etiology and diagnosis of traumatic psychoses. The patients who formed the basis for this study, were selected from among those listed on the hospital records as traumatic psychoses and those admitted during the year 1936-1937 with such a diagnosis or with a history of severe head injury.

The total number of patients studied was thirty-four (34). Twenty-six (26) or seventy-six per cent. (76%) were male and eight (8) or twenty-four per cent. (24%) were female. The preponderance of male over female patients in this group is readily explained by the increased hazards in occupations and activity taken by men and also to some extent by the alcoholic habits of men as compared with women.

The age of the patient was interesting. Two (2) of the patients were between twenty and thirty (20-30) years of age or five per cent. (5%) of the total. Ten (10) or thirty per cent. (30%) were between thirty and forty (30-40) years of age and twenty-two (22) or sixty-five per cent. (65%) were between forty and sixty (40-60) years of age. These findings suggest that in the younger age groups the recuperative powers are better and, except in severe head injuries which result in death, recovery without psychosis is almost certain to occur. Occasionally, mental symptoms suggesting mental enfeeblement may follow such injuries in younger persons. On the other hand, persons in the older age groups are predisposed by vascular changes in the brain and the general lowering of resistance with age to such degrees that trauma sufficiently severe to produce a psychosis in the middle age group probably results in death in the older age group.

As would be expected, injury by or within vehicles was the most common immediate cause of injury to the head. Nineteen (19) patients were injured by or while riding in a fast moving vehicle. All but three (3) of these accidents were caused by automobiles. Two (2) were injured in street car accidents and one (1) in a train wreck. Eleven (11) patients sustained severe injuries to the head during falls. Six (6) of these fell from heights during occupations, such as painting, house cleaning, etc. Five (5) of the eleven (11) fell during an alcoholic debauch. In three (3) patients injury was sustained by objects striking the head and in one (1) case no history as to the nature of the trauma was available.

The relationship of alcoholism to development of traumatic psychoses was rather obvious as a great number of these patients presented a history of excessive drinking. Of the thirty-four (34) patients studied, fourteen (14) were heavy drinkers of alcohol. At least eight (8) of these were injured while inebriated. In one (1) patient, the development of the alcoholic habit occurred after the injury and seemed to be a part of the symptomatology produced by the injury. Twenty (20) patients had a history of abstinence or no history was available as to alcoholic habits. The author felt that alcoholic excess was a definite factor in predisposing individuals receiving head trauma to marked mental disorder.

An example of the relation of alcoholism to head trauma and traumatic psychoses is shown by the following case history: J. D. was admitted to Ward 23 of the Cook County Hospital on September 19, 1936, with a history of severe head trauma having occurred during an auto accident on the same day. The patient had been drinking and was drunk at the time of the accident. There was a history of previous mental symptoms due to alcoholism. A spinal puncture on admission to the County Hospital revealed the presence of blood in the subarachnoid space. On admission to the Chicago State Hospital, the patient was found to be markedly confused and showed marked impairment in the intellectual sphere. A neurological examination revealed a motor weakness of the left side of the body with the left patellar reflex more active than the right. After several months, the patient had shown considerable improvement and was paroled.

The relationship of skull fracture to development of psychoses is, of course, evident in that the severity of lesions producing a skull fracture is often great and therefore is quite likely to be associated with severe brain damage. It was not possible to check all patients with roentgenologic studies of the skull on admission, but of the cases studied, sixteen (16) or fifty per cent. (50%) were reported in the histories to have

sustained a fracture of the skull according to roentgenologic findings.

One (1) patient had definite clinical evidence of a 'basal skull fracture although reported as being negative for evidence of skull fracture. In eleven (11) patients, no mention was made of roentgenological study and there was no definite clinical evidence of skull fracture.

It has been the tendency in the past to lay emphasis upon the presence or absence of skull fracture. Although, as evidenced from the above, skull fracture indicates to some extent the severity of trauma, I wish to emphasize that there may be mild trauma with skull fracture and no mental symptoms, mild trauma with skull fracture and mental symptoms, and finally mild trauma with no skull fracture and no mental symptoms. On the other hand, trauma may be exceedingly severe with no skull fracture and no mental symptoms, or both skull fracture and mental symptoms. The difficulty of roentgenologic diagnosis of basal skull fracture is another factor to be considered in evaluating their importance.

Histories are highly unreliable for determining the period of unconsciousness since the relatives' opinions are the basis for the report in a great number of instances. However, in many cases such reports came from the hospital in which the patient had previously been. Again the factor as to just what constituted the period of unconsciousness was not always definitely delineated clinically.

In fifteen (15) or forty-four percent. (44%) of the cases, a history of unconsciousness of a period exceeding twenty-four hours was available. In many of these cases, the report indicated that many days and even weeks elapsed from the time of injury to the recovering of consciousness. One of these patients had a period of unconsciousness followed by a lucid interval of several days and again a period of unconsciousness. Surgical intervention was indicated and on opening the skull a subdural hematoma was found. In four (4) or twelve per cent. (12%) of the cases, a period of unconsciousness of from a few minutes to several hours was reported and in fifteen (15) or forty-four per cent. (44%), there was no report as to the period of unconsciousness in history. The absence of such a report does not mean that no period of consciousness existed so that the total number who actually suffered such a loss, is probably in excess of that indicated. It is quite evident that a period of prolonged coma is a significant prognostic sign as to the possible development of a traumatic psychosis. It seems self evident that prolonged coma is indicative of severe brain injury which in turn is apt to be followed by persistence of mental symptomatology.

Spinal punctures were not done by the author except in a few instances but the following record of the spinal fluid findings is included in this study for the sake of completeness. The report was obtained from the social service records of the Cook County Hospital which accompanied every patient, or from spinal punctures performed after admission to the state hospital. There were a total of eighteen (18) reports available in the thirty-four (34) patients studied. No reports were available on the other sixteen (16) patients.

No.	Unconscious	Period of Unconscious	Spinal Taken After Injury	Cells	Pandy	R. Jones	Lange and Serology
1.	Yes	Unknown	1 day	38	++	++	0
2.	Yes	Unknown	1 day		NO BLOOD IN SPINAL FLUID		
3.	Yes	Unknown	1 day		BLOODY SPINAL		
4.	Yes	4 days	9 days		XANTHOCHRONIC FLUID		
5.	Yes	1 week	2 wks.	4	0	0	0
6.	Yes	Unknown	2 wks.	0	0	0	0
7.	Yes	sev. days	2 wks.	2	0	0	0
8.	?	?	7 wks.	3	0	0	0
9.	Yes	sev. days	2 mos.	0	0	0	0
10.	Yes	sev. days	4 mos.	14	Trace	Trace	1113321000
11.	Yes	5 days	5 mos.	?	+	+	0
12.	Yes	2 weeks	6 mos.	5	0	0	0
13.	Yes	Unknown	6 mos.	12	+	+	0
14.	No	0	1 yr.	11	0	0	0
15.	?	?	2 yrs.	0	0	0	0
16.	Yes	2 days	5 yrs.	2	Trace	0	0
17.	?	?	5 yrs.	2	0	0	0
18.	Yes	?	11 yrs.	4	0	0	0

The accompanying chart shows the findings in the patients studied. In only four (4) patients, were reports available within two weeks of the date of injury. In two (2) of these the report indicates definite evidence of blood in the spinal fluid. In one patient, although no mention was made of evidence of blood in the spinal fluid, other findings were present suggestive of changes in the central nervous system. In one (1) patient, the report read no blood in the spinal fluid. Although the author would like to avoid definite conclusions based on the findings noted in the chart, nevertheless, one would tend to feel that spinal fluid findings suggestive of trauma, occur shortly after the injury responsible for the development of psychoses but that such findings are reversible.

Occasionally during the classification of patients with a history of injury to the head, a diagnosis of traumatic psychosis is offered in spite of the fact that the injury was sustained many years before the onset of the psychosis. There were four (4) patients, two (2) of whom were not included in this study, whose psychoses developed after a lapse of time of more than several months' duration between the date of injury and the onset of the psychoses. The two (2) patients not included in the group, were classified as traumatic psychoses. However, in both instances, the injury to the head occurred many years before the onset of the disease and the injury itself was described in the histories as apparently of not sufficient severity to cause unconsciousness. The mental symptomatology at the time of admission was that of schizophrenia and the clinical course substantiated the diagnosis of schizophrenia. It was definitely felt that the two (2) patients were mistakenly diagnosed as suffering from a traumatic psychosis. Of the thirty-four (34) patients included in the study, only two (2) could definitely be said to have developed a mental disorder some time after the injury. In one case, the patient developed Jacksonian epilepsy after a lapse of several years following the accident and concomitant with the development of convulsions the patient developed mental symptoms. The other patient suffered a basal skull fracture followed by loss of hearing in both ears and development of paranoid ideology two years after the accident. It would seem inadvisable to venture a diagnosis of traumatic psychosis in an individual whose mental symptomatology first manifests itself some time after the accident.

The frequency with which neurologic findings were noted is not surprising for one would expect an injury sufficiently severe to produce mental changes, would also produce brain damage severe enough to result in permanent neurologic manifestations. The involvement of cranial nerves was most frequently seen in third, fourth and sixth nerve damage. In ten (10) patients, there were found such neurologic findings as unequal pupils, non-light reacting pupils, irregular pupils, strabismus due to rectus muscle weakness and ptosis. Six (6) patients, developed hearing defects following the head trauma and six (6), were reported as having a facial paralysis or weakness. In five (5) patients, dysarthria was noted while there was no mention in the history of any defect in the twelfth nerve, and in three cases, definite paralysis of the hypoglossal nerve was present. In one (1) patient, a paralysis of the spinal accessory nerve existed. Eleven (11) patients, had evidence of pyramidal tract disease manifesting itself in conditions varying from a complete hemiparesis to an inequality of reflexes. Thus twenty-four (24) of the thirty-four (34) patients, had neurologic findings with variations from minor pupillary changes to alternating hemiplegias. The frequency with which neurologic findings accompany the occurrences of a traumatic psychosis suggests that patients showing persistent neurologic findings following the period of unconsciousness, are probably more likely to have a psychosis than those in whom neurologic findings soon reversible disappears.

The mental symptoms noted, varied from an acute delirium, with disorientation, disconnected speech, semi-stupor to coma, followed by amnesia with rapid recovery, to a profound dementia. A fairly large number of the patients showed a Korsakow-like syndrome, with confabulation and fabrication, disorientation and loss of memory for recent events. A dementia simulating the condition seen in mental defectives was not uncommon. Especially interesting were two cases of paranoid type of psychoses in both of which there was impairment of hearing. However, no attempt was made to carefully evaluate the symptoms. The prognosis seemed to be more favorable in patients with an acute delirium. Recovery was uncommon in cases which showed no pronounced improvement in from five to six

months. Death was most likely to occur in patients who remained disturbed and disoriented for some time, and began to show a downhill course due to exhaustion and inanition.

CONCLUSIONS

In a study of thirty-four (34) cases diagnosed traumatic psychoses, the following was concluded:

1. Males are predisposed to head injury, therefore more apt to develop a traumatic psychosis.
2. Individuals of the older age groups are most likely to become psychotic after trauma to the brain.
3. Injuries are more commonly caused by and in vehicles than by any other agent.
4. Alcoholism is frequently an associated factor in head injuries which are followed by mental manifestations.
5. Skull fracture is found in a large percentage of cases, but the injury to the brain and not the cranium is the important factor.
6. A long period of unconsciousness following trauma is presumptive evidence of sufficient brain injury to make the development of a traumatic psychosis more likely.
7. Psychoses which do not appear shortly after head injury are not apt to be traumatic psychoses.
8. Neurologic findings are very common in individuals who, following a head injury, have developed a psychosis.

PSYCHOTIC REACTION FOLLOWING TRAUMA

D. Louis Steinberg, M. D.

ELGIN, ILLINOIS

The importance of head trauma as an etiologic factor in psychotic reaction has long been a debatable question. This may be due to the complexity of factors that must be evaluated in establishing causal relationships. There are numerous reports dealing with so-called post-traumatic neuroses,[13] but there are comparatively few concerned with the role of head trauma as initiating psychotic reactions,[4, 5, 10] or precipitating such syndromes as general paralysis and arteriosclerosis.[2, 3, 11, 12, 13]

In spite of the evident increase in incidence and interest in this subject,[4, 7, 13] as emphasized in the preceding paper, none of the large insurance underwriters contacted had any available statistical data.

A survey of the literature indicates that the majority of the psychotic reactions immediately following trauma are reversible and this has given rise to an impression that the prognosis for post-traumatic psychoses is good.[6, 8] In a mental hospital such as Elgin, however, we receive patients whose acute symptoms of trauma have subsided, but whose psychotic reactions are severe and prolonged enough to necessitate institutionalization.

The histories of the 4,500 patients that comprise the present population of the hospital were reviewed, in addition to checking the statistical files from 1928 to the present, for "post-traumatic" diagnoses. The patients with a definite history of trauma were studied carefully to determine if there was any relationship between the trauma and the development of psychotic symptoms. All those with inadequate histories in which the relationship was questionable—and there were a great many of these—were excluded from our study, as also the cases of psychoneurosis and schizophrenic-like reactions, the latter because they will be reported in the following paper.

The final material for this study consists of 37 subjects—four diagnosed general paralysis, one diagnosed cerebral arteriosclerosis, both with history of head injury—and 32 patients with psychotic reactions initiated by such trauma.

There is no accepted criterion for proving trauma to be a precipitating factor in general paralysis; however, the latency period from time of injury to onset of the symptoms of paresis is thought to be a measure of the importance of the trauma.[2, 3, 12, 13] The similarity between some of the post-traumatic symptoms and those of general paralysis makes an evaluation of the role of trauma extremely difficult in long standing cases such as ours.

From the Elgin State Hospital.

I wish to acknowledge my appreciation to the following: Dr. Charles F. Read, Managing Officer, for his encouragement, suggestions and constructive criticisms; Dr. Erich Liebert, Clinical Director, for suggesting the problem and for his assistance in outlining the approach to the problem; and Phyllis Wittman, Ph.D., for the psychometric studies on these patients.

Read before Physician's Association of Department of Public Welfare, Illinois State Medical Society, May 2, 1939, Rockford.

In a population of 452 patient suffering from paresis, only four classified for our group. Of these, two showed symptoms of paresis immediately after trauma, one six months after and one a year after the injury. These results, showing a probable relationship between trauma and onset of paresis in less than one per cent., corroborates the opinions of Kinier Wilson and Strauss and Savitsky that trauma as a precipitating factor in paresis is negligible.

From 325 patients diagnosed psychosis with cerebral arteriosclerosis, only one patient developed mental symptoms soon after the head trauma.

The 32 cases in which head trauma appeared to be the initiating factor in the development of a psychosis, included the following psychiatric diagnoses: 29 cases of posttraumatic syndrome and one case of Parkinsonian syndrome, one psychosis with mental deficiency and epilepsy, and one case of chronic alcoholism.

In this group of cases there were no reported factors in the heredity or pretraumatic life of the patient which could be interpreted as predisposing the patient to a psychotic reaction following the head trauma.

Three of our cases had sustained more than one severe head injury, but although they showed definite evidence of brain damage after the first trauma, mental symptoms were not severe enough to necessitate institutionalization at that time. Case 1 had a severe head trauma at the age of seven which resulted in mental enfeeblement. At the age of 34 a second accident resulted in a skull fracture, following which he developed a psychosis and was institutionalized one year later, and since then has been showing a progressive dementia.

Case 8 at the age of 42 fell from a roof and following this developed epileptiform seizures. He had a series of three more accidents, the last one at the age of 48, at which time he was committed to the institution. He has continued to have generalized epileptiform seizures and has developed deafness and a marked dementia.

Case 15 had a skull fracture very early in life; treated by decompression. At age of 39 he was struck on the head by a board and then developed epileptiform seizures and mood swings.

Statistical analyses brought out the following data:

Table 1 lists the most frequent causes of head trauma. Falls from a height are most numerous, with automobile accidents a close second.

The other part of this table indicates the type of injury and shows the predominance of recognized skull fractures. It is possible that among those diagnosed as concussions there may have been a few unrecognized skull fractures. This analysis indicates that skull fracture and concussion are of approximately equal significance as far as prognosis is concerned.

The age at time of accident varied from 1½ to 53 years, with the greatest frequency in the 4th decade and greatest number of admissions to the institution during the 5th decade. Table 2 corroborates the opinion held by many authors[1, 8]

TABLE 1

RELATIONSHIPS OF ACCIDENT TYPE AND INJURY TYPE TO PROGNOSIS

	Present Status			
Accident Type	No. Imp.	No. Stat.	No. Det.	% of Total
Fall from Ht.	3	1	6	31%
Auto Accident	4	2	2	25%
Crushing Injury	..	3	1	13%
Gun Wound	..	1	..	3%
Unknown	4	..	5	28%
Total	11	7	14	100%

	Present Status			
Injury Type	No. Imp.	No. Stat.	No. Det.	% of Total
Skull Fracture	4	5	8	54%
Concussion	5	1	5	34%
Severe Bruising	2	..	1	9%
Bullet Wound	..	1	..	3%
Total	11	7	14	100%

TABLE 2

RELATIONSHIP OF AGE DATA TO PROGNOSIS

ACCIDENT AGE

	Present Status			
Age Level	No. Imp.	No. Stat.	No. Det.	% of Total
0- 9 yrs.	1	..	1	6%
10-19 yrs.	1	2	1	12%
20-29 yrs.	2	1	1	12%
30-39 yrs.	3	1	3	23%
40-49 yrs.	4	2	4	32%
50-59 yrs.	..	1	4	15%
Total	11	7	14	100%

ADMISSION AGE

	Present Status			
Age Level	No. Imp.	No. Stat.	No. Det.	% of Total
0- 9 yrs.	..	..	..	..
10-19 yrs.	1	..	..	3%
20-29 yrs.	4	2	1	22%
30-39 yrs.	4	..	2	18%
40-49 yrs.	1	3	3	28%
50-59 yrs.	1	2	8	35%
Total	11	7	14	100%

that the later in life the accident, the poorer the prognosis.

The duration of symptoms is also a factor in the prognosis. The common opinion is that if the symptoms have persisted for two years or more, the prognosis is poor.[5, 13] In our material the majority of the patients were admitted to the institution at least two years after the accident.

The onset of physic symptoms following trauma is most often immediate, but there may be a latent period between recovery from the acute symptoms and onset of psychosis. An analysis of our data shows that in 28 or 87 per cent. there was no recognizable latent period. In three cases or 9 per cent. there was a latent period of three to five months, and in one case, of four years. The preponderance of cases showing a continuation of symptoms makes one suspect that with more adequate histories some evidence of mental symptoms would have been found in the other four cases also during the so-called latent period.

Seven patients or 22 per cent. developed their first epileptiform attack from five months to eight years after the accident, with all the evidence pointing to an organic origin.

The reaction to a severe head trauma is usually, though not necessarily, initiated with a variable period of unconsciousness and follows a regular sequence of delirium, confusion, memory defect, confabulation, etc., on to either apparent recovery or progressive deterioration; or may remain stationary at any level.

Table 3 shows the variability and frequency of the acute reactions. Unconsciousness, delirium and confustion are most frequent. Irritability, uncooperativeness, headaches, convulsions and memory defects are next in frequency. Confabulation was noted only once, which is not in accord with the usually reported[8, 13] sequence of reintegration of brain function following unconsciousness due to head trauma.

One of the most important reasons for institutionalization of a patient is his inability to adjust in his former environment. This is due to some change in personality reaction.

Table 4 gives a differential analysis of the pretraumatic and post-traumatic personality types. Because of the difficulty in many cases of classifying personality under one general heading, several descriptive terms are used, consequently the number of frequencies in this table is greater than the number of patients. It will be noted that there is a marked shift in the personality traits. Before the trauma more than one-half of these patients were considered of social or average personality; after the trauma not a single case could be thus reported. The most frequent post-traumatic personality traits exhibited were irritable, paranoid and anti-social components. There is no striking relationship between personality type and prognosis, but for those patients showing a paranoid post-traumatic reaction the prognosis appears to be poor.

Many authors[9, 13] have emphasized the frequency of subjective complaints of patients following head trauma, such as headaches, dizziness and fatigability. In this group these complaints were relatively infrequent.

All have definite pathologic organic brain lesions. Headache, which is stressed in the liter-

TABLE 3

RELATIONSHIP BETWEEN ACUTE REACTION TYPES AND PROGNOSIS

Acute Reaction Types	Present Status No. Imp.	No. Stat.	No. Det.	Percentage of Total Cases
Unconsciousness	6	3	11	63%
Delirium and Confusion	4	3	11	56%
Personality Defects	4	2	10	49%
Emotional Defects	4	2	6	37%
Memory Defects	3	1	7	34%
Hyperactivity	4	2	3	28%
Muscular Incoordination	..	..	7	22%
Headaches	4	..	3	22%
Habit Loss	..	..	4	12%
Hallucinations	2	..	2	12%
Retardation	1	..	3	12%
Sensory Impairment	..	1	2	9%
Delusions	1	1	2	9%

TABLE 4

RELATIONSHIPS OF PRE-TRAUMATIC AND POST-TRAUMATIC PERSONALITY TO PROGNOSIS

Pre-Traumatic Personality	Present Status No. Imp.	No. Stat.	No. Det.	% of Total
Social	4	3	3	31%
Average	2	1	4	22%
Alcoholic	3	2	2	22%
Asocial	2	..	3	15%
Irritable	1	1	2	12%
Antisocial	..	1	1	6%
Inadequate	..	..	1	3%
Unknown	2	..	3	15%
Post-Traumatic Personality	**Present Status No. Imp.**	**No. Stat.**	**No. Det.**	**% of Total**
Irritable	5	2	5	38%
Antisocial	1	1	6	25%
Paranoid	..	2	5	22%
Asocial	1	1	3	16%
Apathetic	1	1	3	16%
Childish	1	..	2	9%
Hysterical	11	..	..	6%
Alcoholic	..	1	..	3%

ature as a very frequent symptom, is complained of by only 27 per cent.

In the large majority deterioration was present in the emotional, occupational and ethical spheres, as well as in the intellectual field. Forty-seven per cent. expressed delusional ideas, while definite hallucinatory experiences were elicited in only 21 per cent.

Table 5 gives the psychometric indices of deterioration as determined by the Babcock scale. The higher the negative score, the greater the degree of deterioration. A negative score of 3.5 or more is considered of pathological significance. The majority of cases tested on admission when retested for this study showed higher negative scores, indicating the progresive nature of the intellectual deterioration.

Complete neurological examination revealed a variety of pathological reactions. Table 6 shows a predominance of cranial nerve involvement which might be explained by the fact that the head was the site of trauma. Some neurological symptoms were present in all but one case. In a few patients finer neurological tests had to be employed to elicit the evidence for involvement of the neural pathways. In three cases in which the injury involved the frontal bone the clinical signs, both neurological and mental, pointed to a lesion of the frontal lobe.

Many investigators[13] consider alcohol to be a factor directly associated with both incidence and prognosis in head trauma, i. e., the damaging effect of the alcohol makes the brain tissue more susceptible to traumatic injury. Table 7 shows the frequency of alcoholism in our cases and a comparison with their present mental status. Deterioration is as frequent in the non-alcoholic

TABLE 6

FREQUENCY OF NEUROLOGICAL SYMPTOMS

Neurological Findings	Frequencies	Per cent. of Total Cases
Cranial Nerve Involvement	25	76%
Pyramidal Tract Involvement	14	37%
Muscle Tone Impairment	12	43%
Aphasia	2	6%
Convulsion	7	15%
Extra Pyramidal Tract Involvement	3	9%

TABLE 7

A—The Alcoholic Factor as Related to Prognosis.
B—Present Mental Status.

Alcoholic Factor	Present Status No. Imp.	No. Stat.	No. Det.	% of Total
Excessive	5	3	3	34%
Mild and P.	1	2	1	13%
None	4	1	6	34%
Unknown	1	1	4	19%
Total	11	7	14	100%

— A —

Present Mental Status	No. of Cases	% of Total Cases
Recovered	5	15%
Improved	6	20%
Stationary	6	65%
Deteriorating	15	..
Total	32	100%

— B —

TABLE 5

PSYCHOMETRIC FINDINGS

Case No.	On Admission Voc. I. Q.	Det. Index	Time Interval	On Retesting Voc. I. Q.	Det. Index	Present Mental Status
1	95	—0.1	4 yrs.	?	—5.7	Deteriorated
2	90	—3.3	2½ yrs.	94	—4.8	Deteriorated
4	..	..	..	102	—8.7	Deteriorated
6	94	—2.0	6 mos.	94	—0.6	Recovered
7	..	..	..	58	—2.5	Deteriorated
8	Uncooperative		3 yrs.	Uncooperative		Deteriorated
10	70	—3.1	..	..	..	Deteriorated
13	90	—1.3	..	..	..	Stationary
18	..	..	..	51	—5.9	Stationary
19	..	..	..	72	—0.8	Deteriorated
20	88	—4.9	6 yrs.	75	—5.0	Deteriorated
21	..	..	..	107	—2.8	Deteriorated
22	75	—3.0	1½ yrs.	70	—1.5	Improved
23	98	—1.7	5½ yrs.	94	—4.2	Deteriorated
24	..	..	..	106	—7.9	Deteriorated
26	50	—3.0	2½ yrs.	47	—3.5	Improved
27	94	—4.2	5 yrs.	94	—2.9	Improved
28	100	—2.2	..	..	..	Improved
29	112	—3.0	1 mo.	112	—3.9	Stationary
30	90	—2.3	3 yrs.	95	—2.8	Stationary
31	50	—3.0	..	..	..	Deteriorated
32	82	—3.2	2 mos.	82	—2.0	Recovered

group as in the excessive and mild alcoholic groups, but the groups are too small to allow any conclusions to be made.

An analysis of the prognosis for the cases as a whole shows that 65 per cent. appear to be stationary or deteriorating. Of the 35 per cent. considered improved only 15 per cent. were recovered or at least improved sufficiently to be discharged from the hospital. This would indicate that the statistical prognosis for post-traumatic psychosis due to severe head injury should be considered poor.

CONCLUSIONS

1. The importance of head trauma as a precipitating factor in general paralysis and arteriosclerosis appears to be negligible.

2. Cases due to severe concussion without demontrable skull fracture show deterioration as frequently as those with definite skull fracture.

3. The symptoms of prognostic significance for ultimate deterioration appear to be age at time of accident, occurrence of convulsions and paranoid reaction following the trauma.

4. Subjective complaints were relatively infrequent and could not be considered any indication of the amount or severity of the existing organic changes.

5. Head trauma appears to have initiated a definite shift in the most prominently expressed personality components.

6. The most frequent expressions of neurological manifestations were signs of cranial nerve and pyramidal tract involvement, as well as disturbed muscular tonus. In three of the cases the mental syndromes could be related to frontal lobe lesion.

7. Studies of the mental efficiency level in these cases indicate a progressive intellectual deterioration.

8. Analysis of 32 cases indicates a poor prognosis for those cases with prolonged psychotic syndrome following head trauma.

BIBLIOGRAPHY

1. Gotten, N.: Head Trauma; Report of 141 Cases, J. A. M. A. 110: 1727-1730, 1938.

2. Gordon, A.: Delayed Organic Disease of the Nervous System following Trauma, Trans. A. Neur. Assoc. p. 218-224, 1930.

3. Klander, J. V., and Solomon, H. C.: Trauma and Dementia Paralytica, J. A. M. A. 96: 1-7, 1931.

4. Malzberg, B., Ph.D.: A Note on the Rate of First Admissions of Traumatic Psychosis, Psych. Quart. 2: 445, 1937.

5. Mapother, E.: Mental Symptoms Associated with Head Injury; The Psychiatric Aspect, Brit. M. J. 2: 1055-1061, 1937.

6. Pilkington, F.: The Treatment of the Mental After Effects of Head Injury, Irish J. M. Sc. 742-745, Dec. 1937.

7. Ramsay, F. B.: Head Injuries from Highway Accidents, J. Ind. M. A. 31: 332-334, 1938.

8. Symonds, C. P.: Mental Disorders following Head Injuries, Proc. Roy. Soc. Med. 30: 1081-1094, 1937.

9. Strauss, I. and Savitsky, N.: Head Injury; Neurologic and Psychiatric Aspects, Arch. Neur. and Psycht. 31: 893-955, 1934.

10. Ibd.: The Sequelae of Head Injury; The Psychogenic Factor, Am. J. Psycht. 91: 189-202, 1934.

11. Ibd.: The Sequelae of Head Injury, N. Y. S. J. Med. 37: July 1, 1937.

12. Wilson, S. A. Kinnier: Role of Trauma in the Etiology of Organic and Functional Nervous Disease, J. A. M. A. Dec. 29, 1923.

13. Wolfson, I. N.: Psychiatric Aspect of Head Injuries, Psych. Quart. 12: 137-174, 1938.

SCHIZOPHRENIC-LIKE PSYCHOSIS FOLLOWING HEAD INJURIES

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Schizophrenic-like psychosis following head injuries has received little attention, while the role that the head trauma plays in producing the so-called posttraumatic psychosis has been studied extensively. The literature also contains reports on the relationship of general paresis to trauma, the incidence of Parkinson's state following severe head injuries or the development of arteriosclerotic and senile conditions.

In 1934 Schilder,[1] in a study of psychic disturbances after head injuries, concluded that the catatonic symptoms may be a sign of local lesion induced by a severe head trauma and that emotional disturbances toward schizoid, psychopathic and epileptoid trends may result from organic changes due to trauma. Meyer,[2] on the other hand, summarized his experiences by saying the trauma is merely a contributing factor in the production of such states as manic-depressive, catatonic deterioration and paranoid conditions.

The psychoanalysts, especially Freud, Ferenczi and Abraham,[3] have claimed that too much attention is placed on the physical aspect, while important, unconscious forces are neglected. Others have suggested that the trauma may produce profound effects from a psychogenic point of view. The question arises whether the trauma releases a latent schizophrenic process that would have developed in the course of time, or whether

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these cases diagnosed as dementia praecox are not really such, but simply schizophrenic-like pictures. In reference to this problem, Rosanoff et. al.,[4] in his work on the schizophrenias occurring in twins, makes the following comment: "A more detailed scrutiny of the material suggests the possibility of separating out from amongst this so-called schizophrenic psychosis, a large group of cases that seem to occur on a basis of partial decerebration, mainly of traumatic or infectious origin."

This communication is an attempt to determine whether or not those cases of schizophrenia developing after severe head injuries are in any way different from those that come on without severe head trauma.

Out of 2,000 cases of dementia praecox that comprise the resident population of Elgin State Hospital, a large number were found that showed some relationship to a severe head injury, but only those were selected in which the connection between trauma and onset of mental symptoms could not well be discarded. The casual relation between the trauma and psychosis was strongly suggested by the following statements that occurred in some of the histories: "Following the accident he became stubborn, indifferent to relatives, restless, sleepless, took long walks by himself," or "Struck down in a drunken brawl, sustained a skull fracture; was unconscious for many hours; remained dazed and clouded for several days; slowly cleared up, but never returned to normal," or, "He has not been well since his injury; was struck by an auto and sustained a skull fracture; memory noticeably poor."

In all cases the onset of mental symptoms occurred within a few hours to three months after the injury. In eleven there was a variable period of unconsciousness after the trauma. All were diagnosed as dementia praecox. The present study, including a detailed examination of the records, as well as observation and mental examination of these patients, shows that two different kinds of reaction may occur. In one group the picture is unmistakably that of schizophrenia and can in no way be distinguished from that which develops without history of precipitating trauma. The psychosis in these cases is characterized by the cardinal symptoms of schizophrenia, namely, a peculiar alteration in thinking and feeling, with a withdrawal from reality. In the ten patients that comprise this group, five had definite psychopathic tainting in the family stock. Six were unmistakably introverted in personality makeup prior to the onset of psychosis, two were extroverted, and in two the data were not convincing. Five patients became seclusive, indifferent, careless, apathetic and exhibited very little spontaneity. In four, the speech became irrelevant and incoherent. All had feelings of external control, hallucinated actively, and presented delusional ideas of a bizarre nature. These patients now show a picture of hebephrenic deterioration. In spite of the fact that some have received shock treatment, no improvement could be seen in any of these patients. As an example, note the following:

Case 1. Twenty-six-year old, single, white male, admitted to Elgin State Hospital 5/29/26. Mother had dementia praecox; sister was neurotic. He had a grammar school education and worked for several years for an electric company. He was not only interested in radio and electrical apparatus, but showed marked mechanical abilities. Described as a seclusive and head-strong personality. Showed no interest in women. He was struck unconscious in an auto accident at the age of 25. Considered queer ever since. Said he was the son of God and could communicate with the world through radio and electricity. He feared someone was after him. When admitted he hallucinated actively, laughed without cause, spoke of Knights of Columbus being after him. For thirteen years he has continued to be delusional; speaks of electrical transmission draining his power. At times he talks irrelevantly and incoherently. He is alternately angry, laughing, indifferent ,and at times has passed into short episodes of catatonic excitement.

Case 2. Twenty-eight-year old, single, white female admitted 11/22/38. Brother is a schizophrenic. She was gradauted from high school at 16, and had a good work record until 1931, when she received a blow on the head in an auto accident and was unconscious for several hours. Within several months she became paranoid and suspicious; thought she was psychic, that she was being impersonated and involved with dope peddlers and white slavery traffic. At times she was quiet, distracted, and subject to laughing spells. On admission she was sullen, resistive, angry, but for no reason would become pleasant and cheerful for short periods. She expressed ideas similar to those mentioned before and named a certain girl friend as the chief impersonator and persecutor. She was treated with insulin, without result.

In contrast to the above cases, eleven patients did not seem to fit into this series because of a quite different symptomatology. It is true that these eleven cases presented impulsive laughter, apathy, indifference, mannerisms and peculiar, bizarre delusional ideas, but in addition there were symptoms that seemed to be

definitely connected with the head trauma and strongly suggestive of parenchymal damage. For instance, five patients complained of persistent headache, two had a history of epileptiform seizures, two complained of confusion and dizziness, and they all showed impairment of memory and orientation. In contrast to the first group the heredity in these patients showed tainting in two cases only. The prepsychotic personality in seven patients did not present introverted trends; they were at least ambivert, if not extrovert. In the remaining four the data were insufficient. The final outcome in these eight cases did not seem to differ from those presented in the beginning. After several years they leveled off and presented a picture of indifference, laziness and unconcern about surroundings. Three cases have not reached this level yet, even after a period of seven years. The following case reports illustrate the behavior of the patients in this group.

Case 3. Thirty-year-old, single, white male, admitted 6/19/29. Father said to be alcoholic. Third grade education at ten years of age. Truck driver by occupation; was in the St. Charles School for Boys because of chronic truancy. He was shot through the head in April of 1919. Soon thereafter he had two convulsions and several others at intervals since. Since 1933 there has been a marked defect in the various memory spheres and persistent complaints of headaches and dizzy feelings.

He was admitted to Elgin because he was unmanageable. Here he was quiet, careless, manneristic, appeared dazed, complained of hearing women's voices saying nasty things. He experienced body sensations which he referred to as electrical interference. He complained that someone read his mind. Enemies were persecuting him. He was being watched, followed and laughed at. He heard them call him names and saw reference to himself in the newspapers.

During the next ten years he made several attempts to escape from the institution. Usually he has been observed standing about in a dull and apathetic manner; continues to complain of women's voices and states that the sun, moon and stars are influencing him. His speech is irrelevant. He is irritable and profane. No convulsions have been observed while hospitalized.

Case 4. Twenty-seven-year old, single, white male, admitted 7/10/37. Family history negative; grammar school education; good work record; sociable, jolly, enjoyed companionship, and went out frequently with mixed groups. He suffered a skull fracture in January, 1933, and was unconscious seventy-six hours. Soon thereafter he became stubborn, indifferent, restless and sleepless. He blocked in his speech, had laughing spells, was quiet for long periods of time, showed forgetfulness, carelessness of personal appearance, complained of continuous pain in top of his head. At times was out of contact; on other occasions was fearful, had a sense of impending danger, pointed to imaginary things in the room and made faces. On admission to Elgin he was indifferent, showed little energy in the performance of his movements, was manneristic, inattentive and seclusive. Complained of headaches every day. Felt fearful, but couldn't explain why. He heard men's voices, but couldn't tell much about them. Recent and remote memory and calculation were markedly impaired. He appreciated the defect in his memory. During the past two years he has continued to complain of pressure in the back of his head, showed lack of energy, masturbated excessively, was manneristic and utterly indifferent.

COMMENT AND CONCLUSIONS

Out of 2,000 schizophrenics 21 cases showed a rather definite relationship between trauma and the onset of psychotic symptoms. The question whether damage to the brain acts as a releasing factor of an underlying schizophrenic mechanism, or whether the changes themselves are responsible for the symptoms cannot be satisfactorily anwered. It is known that in any organic brain disease schizophrenic symptoms may occur, but conclusive evidence is required to show that these changes are responsible for the mental symptomatology. In our first group of patients there was no evidence of brain injury, but still the mental symptoms developed in such a close time relationship to a severe head trauma that this casual sequence of events cannot be disregarded. These patients were all of introverted personalities and had marked hereditary tainting, so that the possibility of schizophrenic-like reaction was present. Due to the injury, this latent tendency became overt and the psychosis which followed the trauma was that of a so-called schizophrenia. We would conclude that in this group the trauma acted simply as a precipitating factor.

In the second group, comprising 11 cases, evidence of brain damage was present. Confusion, dizziness, defects in memory and orientation, as well as persistent headaches with irritability do not belong commonly in a schizophrenic-like picture. Furthermore, in seven of these cases, the pre-psychotic personality was well integrated and hereditary tainting was present in only two. Aside from the mentioned symptoms, these patients presented a schiozophrenic picture. They were all diagnosed dementia praecox and eight of them showed in the course of time a complete deterioration. Still, the neurological changes, the mixture of organic-like mental symptoms and the absence of any predisposing factors in the history suggest that in these cases the trauma did more than precipitate a psychotic reaction. In producing pathological changes in the brain, the

injury would seem to have contributed to the formation of the clinical picture. Although autopsy findings are as yet not available in this group, the development of Parkinsonian-like syndrome in one case and evidence of hemiplegia and convulsions in another suggests a localized brain damage.

In evaluating the role which the trauma plays in the schizophrenic-like picture, one should always pay attention to the personality as a whole. In all cases of so-called schizophrenia one should consider whether the psychosis is not attributable to some definite etiological factor, such as trauma, infection or tumor. Many studies are now being carried on in cases of schizophrenia. The results are interesting, but certainly of questionable value if the study consists of patients whose psychosis can be attributed to some definite etiological factor. Therefore, the need is emphasized to be more exact in evaluating all mental symptoms, so that these patients may be differentiated from the so-called schizophrenia in which no traceable etiological factor occurs.

BIBLIOGRAPHY

1. Psychic disturbances after head injuries, Amer. Jour. Psychiat. **91**: 155, 1934.
2. Anatomical facts and Clinical Varieties of Traumatic Insanity, Amer. Jour. Insanity, **60**: 373, 1904.
3. Ferenczi, S.; Abraham, K.; Jones, et. al.: War Neurosis and Psychoanalysis, London, International Psycho-Analytical Press, 1921.
4. Etiology of So-Called Schizophrenic Psychosis, Aaron J. Rosanoff, Amer. Jour. Psychiat. **91**: 247, 1934.

DISCUSSION

Dr. John Lindquist: I have been very much interested in this discussion of mental disorders following head trauma. I must confess that my experience has been somewhat limited, since I see more of the acute cases of head injuries. Nevertheless, we are concerned about the eventual fate of these patients and Dr. Mock has often expressed a hope of studying state hospital patients in an attempt to answer this problem by a careful follow-up. This is difficult to do because these patients get away and our follow-up records are often incomplete. We really do not know what the eventual fate of most of them is. If any advances are to be made, they must come from a study carried out in the state institutions.

There are several things from the standpoint of the general surgeon who deals with acute injuries that interest me. First, in regard to the prognostic features that have been pointed out, particularly the age group. Dr. Goldstein's statistics indicated that the older the patient the less likely there is to be a reversible reaction. We have patients who could be called subacute cases of psychosis or neurosis that seem to improve if we dehydrate them or do spinal drainage, and I should like to ask Dr. Goldstein if he has had any experience with this management in chronic cases. Fay of Philadelphia feels that there is cerebral ischemia that may be helped by dehydration or spinal drainage.

Dr. Steinberg's remarks regarding the apparent similarity between the prognostic effects of concussion and serious head injury or skull fracture are interesting. There is a tendency among men who treat acute cases to consider concussion as of minor importance and not to give these patients as adequate treatment as those with serious injury. We have felt that in definite signs of concussion the patient should receive the same care as he does with a serious injury. I should like to ask Dr. Steinberg if there is any correlation between adequacy of treatment and the degrees of traumaic psychosis in his study.

I think much is to be gained from pathologic or postmortem study of these patients because in our management we are often puzzled about indications for encephalography or craniotomy on one side or both sides, and there is some divergence of opinion as to when these should be done.

I was interested in seeing Dr. Shapiro's classification: those with pathologic changes in the head and those without pathologic findings.

I feel a great deal is to be gained in this direction and I feel the psychiatrist has to point the way in regard to the prognosis and diagnosis and treatment.

Dr. T. M. French, Chicago: My experience with this type of case is limited so I shall have to make my remarks somewhat general. I should like to compliment the three essayists for these papers which furnish a very nice survey of this field. The paper which lies nearest to my own field is that of Dr. Shapiro. He, as I understand it, divides schizophrenic-like reactions associated with head trauma into two groups: one in which the head trauma acts as a precipitating agent, in which the organic features are less pronounced and in which the previous personality of the patient would suggest that the condition might have developed without the trauma. The other group has organic features which are much more pronounced and there is less reason to suppose that these patients would have developed a schizophrenia without the head trauma; for that reason he regards them as much more specific cases of head trauma with symptom picture of schizophrenia.

This classification seems to be very useful and suggests certain points that I think are worth emphasizing. Dr. Shapiro ended his paper with an appeal that we should study the detailed symptom complex of these patients with reference to the organic and psychic background, and it is that point which I think deserves being emphasized. It seems to me in cases like his second group it is important to realize that even in such cases where there is very definite organic involvment, the symptom-picture or the clinical picture needs to be analyzed, if we want to understand it, into several factors. In the first place, what are the direct effects of organic damage? In most cases there are memory and intelligence defects. Sometimes they are neurological findings probably attributable to actual

damage to the brain. Such symptoms as irritability, headache, confusion and fatigue are probably very largely to be attributed to the direct psysiologic effect of the damage. In the second place, even in psychoses due to definite organic damage, there are also very important aspects of the clinical picture due to psychic reactions. The most important of these I would say are psychic reactions to the organic damage itself. There have been some interesting studies made along this line, especially one by Ferenczi on paresis, and it would be interesting to apply the same line of thought to traumatic neuroses. Ferenczi and assistants found that if they carefully examined the content of the delusions of a paretic, a large number of them may be best understood as psychic reactions to the existence of the organic defect. A number of their patients reported their age falsely, giving instead the age at which they had acquired their infection, as they did not wish or admit to themselves the fact that their infection had ever occurred. In other cases the character of the delusions of the grandiose type had to do with some attempt to deny the fact of their infection or to deny the fact of the mental impairment that resulted from it. The delusion that they were treating people for syphilis was an attempt to overcome the trauma of realization that they had acquired this despised and disabling disease and instead of that they were picturing themselves as treating thousands of people with that disease.

I recall a case of my own in which the patient developed a manic depressive-like reaction. This was a case of paresis. During the expansive phase he did not remember his infection, but during his depressive periods he was painfully aware of it. Grotjohn has discussed at some length the juvenile paretic from this point of view, with the emphasis upon the reaction of these young children to the tremendous inferiority and feeling of insecurity that resulted from the fact that they were unable to use their minds as they normally should, and he has made some therapeutic suggestions based upon the principle of trying to minimize the traumatic effect of the realization of the mental defect.

Finally, when the patient is exposed to this tremendous trauma and organic damage and to the psychic shock of being disabled, the type of his reaction will depend not only upon the nature of the damage and not only upon the nature of the psychic trauma at the time, but also upon the structure of his personality. The organic damage and the physic reaction to it tend to destroy the integration of the personality and one should be able to find a relationship, not necessarily crude and superficial, but a definite relationship between the actual structure of the personality and the type of reaction to the cerebral insult.

Dr. H. H. Goldstein, Chicago: I am sorry to inform Dr. Lindquist that we did not do any significant number of spinal taps on patients admitted with a diagnosis of traumatic psychosis. I doubt whether there would be any significant change since most cases are admitted to the hospital some time after the trauma, at least several weeks, and although it might be interesting to see what the result would be, we have no information as to what would have taken place.

Dr. D. Louis Steinberg, Elgin (in closing): In answer to Dr. Lundquist's question, when we prepared this study we were careful to use only material we were familiar with. The histories as a rule were too inadequate to make any correlation of the patient's present condition with treatment received immediately following the injury. We do get some histories that give adequate information regarding the injury, what was done and the condition of the patient at the time of discharge, but most of the histories were not adequate and consequently we could not correlate the treatment to the outcome.

Dr. Louis B. Shapiro, Elgin (in closing): I think it is important to state that in going over the patients who are in Elgin, many were found who received injuries to other parts of the body who reacted in a schizophrenic-like manner. For our study we chose those cases in which the injury was to the head and of a severe degree. It is quite obvious that many patients of introvert make-up may respond to a trauma from a psychic point of view.

This study is being continued, as suggested by Dr. French. Unfortunately many of these records are inadequate. We are, however, contacting the relatives of patients whose psychosis is causally related to trauma with the purpose of obtaining detailed anamnestic material relative to his personality and his psychic reaction to the trauma.

ACUTE SUPPURATIVE OTITIS MEDIA AND MASTOIDITIS

M. A. Glatt, M. D., F. A. C. S.

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Acute suppurative otitis media and mastoiditis are usually secondary to an infection of the upper respiratory tract by the streptococcus pneumococcus, influenza bacillus and other bacteria. These bacterial infections and the associated systemic diseases, such as scarlet fever, measles, influenza, pneumonia, diphtheria, may often produce variations in the course of the disease.

In order to clearly understand the symptoms, which may arise during the early or late course of the disease, it is advisable to remember that they are the effect of either irritation or destruction of the various structures in or around the temporal bone. Those structures are the mastoid air cells, lateral sinus, dura, labyrinth, facial nerve in the tympanic and mastoid portions; the trigeminal and abducens nerve, bone

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marrow and air spaces in the apical portion of the petrous pyramid.

The type of pneumatization of the mastoid process may alse be a factor in the variability of the symptoms. The pneumatic type of a mastoid process differs from the diploic type in that its cortex is usually thinner, the air cells are larger, less vascular and have less absorbing abilities. The sclerotic type has an extremely thick cortex and contains few or no cells. Because of their difference in structure these mastoid processes when infected may often present a different clinical course.

SYMPTOMS OF OTITIS MEDIA AND MASTOIDITIS

The infection, as is known, extends to the middle-ear by way of the eustachian tube. The mucosa of the mastoid antrum, being continuous with that of the middle-ear, becomes infected at the same time.

With the exception of the fulminating cases, where a complication arises coincidental with the ear infection, there is usually a period of localization in the middle-ear and in the mastoid antrum.

There is a complaint of earache, mild fever and deafness. This is accompanied by the various progressive changes in the ear drum, such as injection, bulging, loss of luster and landmarks. Further extension of the process may give rise to the following more or less severe and alarming symptoms:

1. The pain may become boring in character and be referred to the entire head. This may be associated with extreme restlessness and loss of sleep. Occasionally, due to irritation of the gasserian ganglion, the pain is felt in the back of the eye, in the face and teeth. While it is an important symptom of petrositis it does not always indicate an involvement of the petrous apex.

2. Tenderness may be elicited at various points of the mastoid process. This is especially true in the pneumatic type of a mastoid process. In infants, due to the incomplete closure of the tympanic ring, an edema may be noticed in front and above the auricle.

3. Fever, usually around 101 and 102; at times, especially in children, may reach 104 and 105, and in diploic mastoids may be associated with chills.

4. Deafness may be extreme by air conduction. The bone conduction, however, may be increased.

5. Dizziness may be complained of. This is due to absorption of toxic substances by the labyrinth or to pressure on its windows. Nystagmus to the same or the other side may be present. The latter is not to be considered as an invasion of the labyrinth in the presence of good bone conduction. Labyrinth irritation is more often found in the diploric or sclerotic types of mastoid processes.

6. Vomiting may be extreme in nature and, at times, in children is associated with rigidity of the neck. This may be due to the irritation of the dura at the nonobliterated petrosquamous suture, or to irritation of the labyrinth.

7. Facial nerve paralysis may be an early finding. It is due to the pressure on the exposed nerve at an area where there is a dehiscence in the bony canal which protects the nerve in its course through the middle-ear. This anomaly is more often found in the well pneumatized temporal bones.

8. Abducens nerve paralysis may at times occur during the early course of the infection. It does not always signify an actual involvement of the petrous apex.

9. The blood picture shows a leucocytosis with an increase in the polymorphonuclears. A positive blood culture may be found early especially in the diploic type of mastoids. It does not necessarily signify a sinus thrombosis, especially if baeteria other than the streptococcus hemolyticus, or the pneumococcus type III are found.

It is remarkable, that those symptoms may subside, some quickly, some gradually, following the spontaneous rupture or after the incision of the tympanic membrane. What seemingly appears as a case of mastoiditis or of another complication may result in a complete recovery. This is explained by the observation that the symptoms, which may arise in the early course of the disease, may be produced by pressure on the surrounding structures by the congestion of the mucous membrane of the middle-ear or of the mastoid cells, or due to absorption of the toxic products of the bacterial infection. Those symptoms do not necessarily signify an actual destruction of tissues or organs.

When, however, some of the above enumerated symptoms persist or become more severe, or ap-

pear after the establishment of drainage from the middle-ear, then one must be on guard to evaluate each symptom with reference to a threatened complication.

DIAGNOSIS OF SURGICAL MASTOIDITIS

It has been observed that the pneumatic type of a mastoid process breaks down much earlier and oftener than the diploic type and forms a coalescent abscess. It usually takes about three weeks from the onset of the ear infection for such a process to occur. Coincidental with it there is an infiltration of the periosteum of the mastoid process and of the bony auditory canal. This produces more or less bulging of the auricle in a forward and downward direction and sagging of the posterosuperior external canal wall. The latter is almost a constant finding in all types of pneumatized mastoid processes and constitutes a pathognomonic sign of surgical mastoiditis.

Retraction and bulging of the auricle is more often found in the pneumatic type of a mastoid process or when there is a breakdown of the external cortex in either type. Although it is a pathognomonic sign, it may be too dangerous to wait for it to appear.

The roentgenogram, which in the early stages of the infection shows a cloudiness of the cells may not always show the corresponding degree of the actual destruction of the mastoid air cells. It is, therefore, not always dependable. However, when at any period during the infection, it shows progressive evidence of destruction or a coalescence of the mastoid air cells, it supersedes all as a pathognomonic sign in the diagnosis of surgical mastoiditis.

There is not always a cessation of the ear discharge with the onset of the symptoms of surgical mastoiditis. It may be unchanged, improved, or increased in the presence of a complication.

Some of the symptoms, such as mastoid tenderness, fever and chills, headache, vomiting, dizziness, facial or abducens nerve paralysis, may have made their first appearance at this stage; some may have recurred with a greater severity after a period of improvement. Each of those symptoms, alone or combined, are at this period significant of the destructive process which is taking place and, in conjunction with the aforementioned otoscopic and laboratory findings, constitutes a definite indication for the surgical mastoid operation.

Occasionally, because of the presence of the pathognomonic signs of destruction of the air cells in the mastoid process, the indications for the mastoid operation may arise apparently very early, i. e., in the first or second week from the onset of the ear discharge. Those are the cases, where because of the severity of the original disease, there has been a delay in the timely recognition of the ear symptoms. Another contributing factor are certain bacteria, notably the pneumococcus type III which promotes the formation of fibrin and granulation tissue instead of pus. Although it is often destructive in nature, it may at first produce mild symptoms. The ear drum may not show the redness and bulging which accompanies the usual case of acute suppurative otitis media and, therefore, does not rupture, or there may be scanty or no drainage even after a long delayed incision.

There are also instances when the absolute indication for the mastoid operation may arise regardless of the absence of the aforementioned pathognomonic signs. Among those are a group of cases, where sepsis with a positive blood culture of the streptococcus hemolyticus or pneumococcus type III, labyrinth and meningeal irritation, and motor nerve paresis continue with a progressively increasing severity in spite of drainage from the middle-ear.

An ear infection cannot be considered cured unless the general symptoms have subsided, the ear discharge had ceased, the ear drum had healed, and the hearing had returned to within its former level.

The usual duration of the discharge is from one to four weeks. In undernourished individuals it may last up to six weeks. A careful search should be made for any constitutional dyscrasia or for foci of infection in the nose and nasopharynx.

The persistency of the discharge after the removal of local foci of infection is indicative of suppuration of some of the mastoid air cells. Because of its detrimental effect on the hearing and the possibility of its becoming a chronic process it presents a relative indication for the simple mastoid operation.

TREATMENT

An incision of the tympanic membrane should be performed when there is a moderate amount

of bulging, associated with fever, restlessness, pain and impairment of hearing. This is especially important in the exanthematous diseases. Marked bulging, especially, in the flaccid portion of the tympanic membrane with impairment of hearing, even in the absence of severe constitutional symptoms, calls for a liberation of the exudate in the middle-ear.

In this connection I may call your attention to a frequent occurrence of a spontaneous rupture of the ear drum, in less than twenty-four hours, after the first examination of it when it appeared only slightly injected.

This demonstrates the occasional rapidity of the process and the harmlessness of early drainage. Having encountered an instance in the prerupture stage, and especially in the presence of one or more constitutional symptoms, one is justified in doing an early incision of the ear drum. In other words, it is the general symptoms and local findings which shall be the guide and not the time element. The exception to this rule is that type of otitis media which is characterized by the formation of hemorrhagic blisters on the ear drum and external canal wall. These may show the hemolytic streptococcus on smear and culture. The administration of sulfanilamide is advisable in these cases. The incision of the ear drum may be delayed for twenty-four hours depending on the signs and symptoms.

The after management consists of the removal of the secretions from the middle-ear and the building up the general resistance of the patient.

The use of mass suction in the treatment of acute suppurative otitis media has no value. While this procedure may have a favorable effect on the chronic discharging ears, in the acute cases, however, such treatment may produce a blocking to secretions and may convert a seemingly mild case into a surgical one. These cases are referred to in the literature as "suction mastoiditis."

It is remarkable how by the use of cotton drains, properly fashioned and frequently replaced, one may remove the secretions. Often, long tenacious plugs of mucopus are found adherent to them.

Irrigation of the ear canal, may be resorted to when there are accumulations of detritus in the external canal which block drainage. There is no proof that this procedure has caused a spread of the infection to the mastoid air cells.

Regarding the use of sulfanilamide, which has been highly acclaimed as a valuable drug in ear infections, I feel that a warning must be given that this drug may often confuse the clinical picture by masking certain symptoms or by giving a false sense of security in converting an active process into a latent form. One must, therefore, use this drug only as an adjunct and not as a substitute for the indicated surgical procedures.

It is not my intention to discuss the details of the technic of the mastoid operation. I only wish to emphasize the importance of adopting a surgical procedure which will exenterate all the accessible air cells around the lateral sinus, dura, labyrinth, zygoma and tip. Thus, numerous instances of complications which may be traced to incomplete surgical procedures will be eliminated.

It is not within the scope of this paper to discuss the complication, known as petrositis. I have only intimated some of the clinical symptoms which may arise when the petrous apex becomes involved in the early stages of the ear infection. Although this area is not explored during the simple mastoid operation, there are instances of petrositis in the experience of others and my own, which have cleared up after a most thorough exenteration of the cells in the simple mastoid operation. One may, therefore, infer that such a thorough procedure may also carry with it an element of prophylaxis in the prevention of petrositis.

The following case report does not represent the usual type of an ear infection, nor an isolated incident. Because of its recent occurrence and its illustration of many points of this presentation, I consider it worth while to cite it.

A white male, aged 28, registered on my service at the Illinois Eye and Ear Infirmary on April 8, 1939, because of his complaint of right side deafness of two months' duration.

He stated that two months previously he had a right side earache which lasted for one week. After a relief of ten days' duration there was an onset of right side facial paralysis. This was followed two days later by an attack of dizziness and vomiting. At that time the deafness was increased to an extreme degree. A diagnosis of Bell's palsy was made by his physician. After a two weeks' rest his condition was improved sufficiently to allow him to resume his usual occupation. He stated that at no time did he notice a discharge from that ear.

At the time of examination the facial nerve paralysis, which was of five weeks' duration, had almost com-

pletely recovered. There was a sustained spontaneous nystagmus by gazing to either side. The ear drum did not present any abnormalities. There was no edema or tenderness over the mastoid process. The tests disclosed a compelte loss of cochlear and vestibular function of the right labyrinth.

Because of his apparent good general condition the patient was not hospitalized by the resident. However, blood and roentgen ray studies of the mastoids were ordered and the patient was instructed to return in two days for a report on the findings. He failed to keep his appointment. One week later information was received that four days after his visit to the clinic he became severely ill. He was removed to another hospital where a diagnosis of meningitis was made.

COMMENT

1. This is possibly an instance of an ear infection with the pneumococcus type III, also known as streptococcus mucosus capsulatus. Since there was no rupture of the tympanic membrane and the symptoms were not severe enough, he did not consult a physician.

2. Facial nerve paralysis coming on during the third week of otitis media with persistent deafness should have aroused suspicion that the condition was not a simple Bell's palsy in spite of the lack of a discharging ear. At this period a roentgen ray study of the mastoid would have already shown destructive changes of the cells. The absence of mastoid tenderness could have been explained by the diploic type of cells which it consisted of and its thick cortex.

3. The onset of signs of labyrinth irritation, such as vomiting, dizziness, nystagmus and profound deafness associated with a history of earache were again sufficient reasons, even in the absence of destruction of the mastoid cells, to explore the mastoid process and more so when there was substantiating proof of a destruction of the mastoid air cells, which by April 8, showed a complete coalescence of all cells.

4. One may avoid many pitfalls in practice by keeping constantly in mind that an ear and mastoid infection have variabilities including the symptoms and local findings and that there is always danger when there is no return of function.

SUMMARY AND CONCLUSIONS

In the diagnosis and treatment of acute suppurative otitis media and mastoiditis it is important to consider each anatomic structure of the temporal bone as a factor in the course of the infection. The symptoms which arise may be a manifestation of either toxic reactions, irritation or actual invasion of those structures.

In most instances the pathologic process is characterized by two stages; one, that of congestion and another that of destruction. The first stage may either subside or progress to the other in a slow or a rapid manner. While both stages apparently present similar clinical symptoms, they can nevertheless, be differentiated.

An incision of the tympanic membrane for drainage of the middle-ear infection is indicated whenever the local findings or the general symptoms are severe in nature irrespective of the duration of the infection.

While the time element is an important factor in the progress from a middle-ear infection to a surgical mastoiditis, nevertheless, one should not adopt this as a strict guide. The indication for the simple mastoid operation arises whenever the pathognomonic signs of destruction of the mastoid air cells present themselves, or when the irritation of the vital anatomic structures increase in severity and threatens the invasion of same.

Due consideration should be given to the use of sero- and chemotherapy before the destructive process has set in. Great caution, however, must be exercised in their use when definite surgical indications arise, lest an active process be converted into a dangerously latent form.

It is important, that the surgical procedures should consist of a thorough exenteration of all accessible diseased cells and not be limited to mere drainage of the mastoid antrum. One may thus clear away the infection from many of the preformed pathways and prevent the formation of new pathways for the extension of the infection to the intracranial structures.

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CLINICAL ROENTGENOGRAPHIC ASPECTS OF PETROSITIS

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Lillie[1] recently wrote "The good that has come from the prominence given in otologic literature to suppuration of the petrous pyramid has risen not from the description of special surgical technics but from the fact that otologists have become more alert than formerly they were to the recognition of clinical syndromes and that they

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more clearly understand the pathologic processes that are associated with the condition." Ballance made the statement that the petrous bone now occupies somewhat the position which the mastod occupied 20 or 30 years ago.

In the United States prior to 1930 little was written or known on the pathology, symptoms, diagnosis and therapy of petrous apex suppuration when Kopetzky and Almour began their serial papers on the subject. The diagnosis of this condition was rarely made except at postmortem. Now otologists and radiologists are on the lookout for this disease entity, for early diagnosis and treatment of this serious pathologic lesion may prevent a fatality. Since operative indications may be questionable every diagnostic aid must be employed.

This paper is an attempt to note what dependent correlation exists between roentgenograhic findings in the petrous portion of the temporal bone and clinical symptoms. The otologist is at times confronted with an ear infection either pre- or post- mastoidectomy wherein uncertain sepsis continues and wherein the diagnosis of petrositis enters. When the recognized characteristic symptom complex of petrositis is present the diagnosis may be evident clinically, but even then the otologist is anxious for more definite information to assure his decision. To what extent do radiographic findings add to such assurance?

As early emphasized by Taylor[2] a complete study of infection in the temporal bone includes proper roentgenographic exposures of this area and its proper interpretation. The petrous pyramid is difficult to study by x-ray because of its depth within the skull and because of variations in its own anatomy. Structurally, the petrous portion of the temporal bone may be pneumatic or diploeic. Either type can be recognized on the roentgenogram. In accordance with Wittmaack's theory if there is no disturbance in the process of pneumatization, a well pneumatized mastoid will result in numerous cases in pneumatization of the petrous portion of the temporal bone. Just as the mastoid process may vary in its structure so may the petrous pyramid and its apex vary. While it is the rule for the mastoid process to be more pneumatic than the petrous pyramid and the apex, nevertheless the reverse condition sometimes exists, that is, the mastoid process is diploeic while the paralabyrinthine cells and the cells at the apex are pneumatic. At times there may be as few as one or two cells at the apex. Mellinger[3] points out that in the interpretation of roentgenograms one must bear in mind the possibility of rather large venous spaces along the carotid canal, which might be mistaken for air cells. Their walls are more irregular in outline than are those of pneumatic cells. Also, the density of osseous tissue increases with age, therefore, one must remember that the bones of children are more transparent than those of adults. These anatomic variations play an important role in the spread of infection from the middle-ear to the apex.

Radiologically, the petrous pyramid in any acute or chronic otitic infection shows a change from the normal. It is known that in the presence of acute otitis media the x-ray film of a pneumatized mastoid process shows a cloudiness. This is due not to an infection in the mastoid process but to congestion and edema of the cell lining membrane which are secondary to and accompany the otitic infection. Likewise the roentgenogram of a pneumatized petrous pyramid in the presence of acute otitis media shows a cloudiness which is due to the congestion and edema of the lining membrane. This is not due to an infection in the pyramid. The petrous area displays a haziness in the presence of acute coalescent or destructive mastoiditis even though there is no extension of the infection into the pyramid. This diminution in aeration is proportionate to the congestion and edema of the membrane lining the air spaces. It is therefore seen that the petrosa shows essentially the same roentgen findings in acute otitis media as in acute coalescent mastoiditis. If suppuration in the petrous apex develops, a marked decrease in density ensuses with halisteresis, softening or destruction of trabeculae, and finally a partial or complete loss of apical contour. A striking demarcation between the apex and labyrinthine portion now becomes evident.

Even with stereoscopic technic it is often difficult in the petrosa to separate one shadow from another and to evaluate the density and outline of each. This is partly due to the fact that most of the petrosal pyramid lies rather deep in the skull; and it is almost impossible to obtain a clear picture of its entirety without the superimposition of the shadows of the sinuses, rim of the orbits, or tissues of the neck and cal-

varium. The main difficulty is inherent in the bone itself. The most common type of petrosa is pneumatized only in a few areas. The surrounding bone is more or less densely trabeculated bone containing marrow. The shadow of this sclerotic or diploeic bone as the case may be is superimposed on the few pneumatic cells and if they are small or located in certain areas their shadows may be completely obliterated.

While obliteration of pneumatic cells in the mastoid usually means pathological changes, this does not hold when considering the petrous pyramid. The more highly pneumatized the petrosa the more the problem of estimating these changes approaches that of the mastoid.

Taylor[4] aptly interprets films with two descriptive phrases (1) an increase in density and (2) a decrease in density. An increase means that a structure appears more white in the roentgenogram. It has gained something. In a pneumatic bone anything that decreases the contact with air produces an increase in density. This may be accomplished by congestion of the mucous membrane, by a transudant or an exudate in the air space, or also by the development of new bone and increase in lime salts. The reverse holds true when a decrease in density is described. Something has been removed, usually calcium and its supporting structure. The area involved appears darker on the film.

By observation of these two changes and correlation of the clinical findings Taylor claims it is possible to diagnose (1) acute coalescent petrositis with inadequate drainage which requires immediate surgical intervention. Here roentgen study shows a diminution in the aeration of the pyramid which is particularly marked in the basal portion. The trabeculae in the apex are thin and decalcified. As the lesion progresses, the halesteresis becomes more marked followed by destructive changes. The decalcification may be so intense that the apex is not visualized in the film. (2) Subacute case of coalescent petrositis, with adequate drainage not requiring immediate operation for drainage of the petrous apex. The same roentgen changes are seen as in acute coalescent petrositis with inadequate drainage but the typical symptom-complex is absent. Otorrhea is present. These cases have a tendency to become chronic. (3) Chronic cases, presenting persistent otorrhea due to an infection in the petrous apex and showing roentgen evidences of chronic osteitis. There may or may not be complete obliteration of all air cells.

To visualize the petrous pyramid either the Taylor or Stenver methods are employed. The mentovertical or so called Taylor base position is the more popular position for the study of the surgical petrous apex. It has many advantages as Taylor has pointed out, the most important being that it allows an over-all view of both petrous pyramids and therefore a comparison between the two sides. The disadvantage is that the projection superimposes the inferior portions of the petrosa upon the superior angle which is frequently involved. The oblique view or Stenvers position gives a good view of the superior angle but necessitates the taking of two plates for comparison of the two sides. The use of a contrast medium as iodochloral when feasible is of great value. Exact technical factors should be maintained so that subsequent films can be reproduced with the same technic, thereby creating a more exact interpretation of changes.

Because of the complexity and gravity of petrositis it seems advisable in a suspected case to take stereoscopic projections by Taylor's method for comparison of both sides and others by Stenver's method for further localization. Today otologists being petrous-minded are inclined in every mastoid case to request films also of the petrosa as a key picture for later study and comparison should the question of petrositis arise. If on the film taken at the outset there is a shadow of either apex no definite conclusion can be made, but if subsequent base plates show a definite change in this area when compared to the original film, then definite evidence of involvement in this area is present.

Considering the barrage of papers and discussions on the subject of petrous suppuration from 1930 to 1935 there has since been only sporadic articles on this condition, particularly the roentgen value. Sufficient time has now elapsed for various observers to report series of cases, and the findings and conclusions in many of these do not correspond with the earlier observations.

Coates and Ersner[5] in 1934 reported four cases each of which showed definite roentgenographic evidence of involvement of the apex of the petrous portion of the temporal bone and all recovered without surgery on the apex, just from myringotomy or mastoidectomy. This brings to

our attention the now well recognized opinion *that for diagnosis of petrositis x-ray films should* never be considered alone but only in conjunction with all other findings. It is apparent that x-ray changes in the petrosa do not necessarily mean operative petrositis any more than x-ray changes in the mastoid necessarily mean operative mastoiditis.

According to Almour[6] the findings on the x-ray films should be interpreted first, as an individual factor giving information of the status quo, and second in comparison with films previously taken of the same patient, affording information of the moving pathological findings. Where only clouding is present and where halisteresis and cell coalescence is absent, and in successive films this status is maintained, one can expect recession of the lesion providing these findings are in accord with clinical observations. Where some evidence of beginning halisteresis or even localized destruction of bone septa is present, but upon successive plates this has not increased, recession of the lesion can be looked for when this finding is associated with gradual improvement in the clinical picture. By this is meant a lessening in the intensity of the retro-orbital pain, a diminution of the otorrhea, and a marked improvement in all symptoms pertaining to the neighborhood structures that may be involved by a petrosal lesion. Where successive x-ray plates show an increase in halisteresis, septal destruction and coalescence and continuation or aggravation of clinical symptoms a progression of the lesion is to be expected and adequate therapy instituted before the suppurative focus ruptures extrapetrosally.

On the other hand Moorehead and Baker[7] consider roentgen evidence of value only to confirm the clinical diagnosis. To them it is the least valuable and least reliable of all signs in determining what process is taking place in the pyramid and what is necessary to do for it. Many times films taken in connection with acute mastoiditis show cloudiness of the pyramid without any signs or symptoms accompanying the condition. In cases in which symptoms of petrous involvement have developed, the roentgenogram may show only generalized clouding without apparent destruction of the cellular partitions. They therefore use x-ray films only in a confirmatory way, if other symptoms are present, and disregard their evidence if it does not coincide with the clinical diagnosis. They, however, *believe that roentgenograms should be taken* stereoscopically with the patient both in the Stenver and Taylor positions. In their cases many of the petrous films were inconclusive or failed to indicate destructive changes in the pyramid in positive operative findings. Of interest in one case roentgenogram of patient's petrosa taken three years after the petrous operation showed no evidence of the previous suppurative process and both sides appear approximately alike on the films despite the extensive necrosis which had taken place.

Williams[8] in his analysis of 30 cases of petrositis from the Mayo Clinic gives little mention to the value of roentgen pictures, indicating main reliance placed on clinical evidence.

Myerson[9] concludes that roentgen study in the two positions outlined above, along with combined symptoms, as the most important single guide. Such study frequently furnishes an understanding of the cellular structure of the petrous pyramid which is essential to an understanding of the disease, and also may indicate cellular channels leading from the middle-ear and mastoid cavity toward the apex tip. The greatest number of fistulae are found anterior and posterior to the superior canal, while the next in number are those located in relation to the orifice of the eustachian tube. Fowler[10] attributes the limitations of roengen examination of the petrous pyramid chiefly due to the exceeding difficulty of interpreting the films. Kopetzky[11] stated that roentgenograms are the only means by which we can determine the advancement of decalcification. Secondary and subsequent x-rays determine its progress and add diagnostic data.

And so as one reviews the papers of the various otologists who have had rather wide experience with cases of petrositis, one finds conflicting and confusing views in their interpretation of roentgenograms to degree of pathological processes and in diagnosis. In the final analysis proper interpretation of films of the petrosal pyramid in conjunction with clinical symptoms depends on the knowledge and experience of both the otologist and roentgenologist as a cooperative team. Frank cases of petrositis requiring surgery are not commonly encountered by the general otolaryngologist but he should be adequately informed of the possibilities and pitfalls

of roentgen study of the petrosa as an adjunct to the diagnosis of a petrosal lesion. To obtain adequate experience in the interpretation of films of the petrosa by both the otologist and radiologist, a base plate should be taken routinely in all mastoid cases as a key film, and later when more detailed information is wanted then both the Taylor and Stenver positions should be taken and repeated at intervals for study of comparative changes as clinical symptoms warrant. At present few hospitals and institutions have established such a routine procedure. In personal interviews with colleagues associated with large hospitals and institutions in Chicago, it appears that the experience with roentgenograms of the petrous pyramid and with frank operative petrous cases is very limited with most of them. It may be possible that with the promiscuous use of sulphanilamide and neoprontosil in otitic sepsis both by the general physician and otolaryngologist the number of mastoid operations has been greatly reduced and thereby greatly diminished the development of petrous cases.

SUMMARY

1. The petrous pyramid is difficult to study by x-ray because of its depth within the skull and because of variations in its own anatomy.

2. The petrosa shows essentially the same roentgen findings in acute otitis media as in acute coalescent mastoiditis.

3. While obliteration of pneumatic cells in the mastoid indicates pathological manifestations such does not apply in the case of the petrous pyramid.

4. For more detailed information exposures of the petrosa should be made in both the Taylor and Stenvers positions.

5. X-ray evidence of lesions in the petrosa does not necessarily mean operative petrositis.

6. Roentgenogram of the petrous pyramid is of value only if properly interpreted in correlation with existing clinical symptoms; and a definite radiographic diagnosis of petrositis should never be made from one film or in the absence of clinical symptoms.

7. To obtain adequate experience in the interpretation of films of the petrosa by both the otologist and roentgenologist, a base plate should be taken routinely in all mastoid cases as a key film and later when more detailed information is desired then both the Taylor and Stenver positions should be taken and repeated at intervals for comparative changes should clinical evidence warrant.

8. At present interpretation of roentgenograms of the petrosal pyramid in otitic sepsis is still somewhat confusing to many otologists and roentgenologists, but the experience that will be gained with routine key films together with future improved technic thereby will in time make the x-ray film of the petrosa a more conclusive adjunct to the diagnosis of a petrosal lesion.

BIBLIOGRAPHY

1. Lillie, H. I.: Arch. Otol. **29**: 345, 1939.
2. Taylor, H. K.: Arch. Otol. **18**: 47, 1933.
3. Mellinger, W. J.: Arch. Otol. **29**: 498, 1939.
4. Taylor, H. K.: Arch. Otol. **22**: 407, 1935.
5. Coates, G. etc.: Arch. Otol. **20**: 615, 1934.
6. Almour, R.: N. Y. State J. Med. **37**: 1495, 1937.
7. Moorehead, R. L., Baker, J. P.: **Arch. Otol. 28**: 499, 1938.
8. Williams, H. L.: Proc. Mayo Clinic Staff **11**: 493, 1936.
9. Myerson, C. M.: Arch. Otol. **22**: 62, 1935.
10. Fowler, E. P.: Ann. Otol., Rhin. and Laryng. **44**: 1056, 1935.
11. Kopetzky, S. J.: Ann. Otol., Rhin. and Laryng. **44**: 1140, 1935.

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DISCUSSION

Dr. George T. Jordan, Chicago: I feel that we all agree that this paper of Dr. Glatt's has set forth in a clear and concise manner the involved anatomy, the etiology and symptomatology and diagnosis of this form of ear trouble with which all otologists so frequently meet.

In the treatment, however, are clear cut cases with the classical symptoms which require a simple mastoidectomy and there are equally clear cut cases which do not require this operation. Then there are the puzzling cases over which we worry; should we, or should we not operate?—the patient with the profusely discharging ear, with all the post auricular tenderness, pain in the side of the head, a pronounced white count, a low grade temperature and perhaps a sagging of the posterior superior wall. We watch this patient from morning till night, and from night to morning, and with palliative treatment he makes a complete recovery. Or perhaps he does not make the recovery and in a week the operation is performed, or all symptoms subside, the patient is normal except a discharging ear and six weeks later a mastoidectomy is deemed necessary to prevent the chronic running ear.

Dr. Glatt's discussion on the treatment has clarified some of these points and aids in arriving at a decision as to just when the operation is indicated.

Dr. George J. Musgrave, Chicago: Dr. Glatt's many years of devoted teaching at Loyola University and in his clinic at the Illinois Eye and Ear Infirmary necessarily gives great authority to his pronounce-

ments. There is no question but that his paper is comprehensive.

Dr. Glatt spoke at some length of the indications for incising the membrana tympani during the course of an otitis media. Though I know that in this room there are two of the world's greatest otologists, I venture to offer a suggestion based on my own experience. For many years I have had a certain conception of what is going on in the middle-ear or in any nasal accessory space before a suppurative process can ensue. The first three or four days of an otalgia indicates definitely a vacuum only, not an infection of the painful area, and certainly there is no pus formation at this stage. Therefore, our entire attention should be concentrated on overcoming the nasopharyngeal congestion and removing, if necessary, the mucopurulent secretion blocking the throat end of the eustachian tube. This should be done regardless of the exact pathology active in the upper respiratory tract. If these procedures prove unsuccessful, then I resort to tubal inflation by the Dench method. Even after pus formation, a satisfactory result is often obtained by establishing drainage through this natural pathway. Many times this plan of treatment obviates the necessity for incision and later major surgery.

The foregoing is offered merely as a suggestion in the treatment, not as a criticism of indicated incision of the drum membrane.

Dr. Gerhard Danelius, Chicago: I wish to limit the scope of my discussion strictly to one problem.

In publications on petrositis, the great majority of roentgenologic illustrations are pictures of adults, while all of us know that the disease affects most frequently young children. The truth is that the taking of satisfactory films of the petrous bone in children is difficult and time consuming. You will greatly help your roentgenologist in prescribing for the x-ray examination a strong sedative (a sufficient dose of sodium amytal—better avertine). The possible damage of a sedative to the infant will be less than the damage of an exhausting fight of twenty to thirty minutes' duration of your little patient with the personnel in the x-ray room.

What roentgenologic technic should be used for a key plate of the os petrosum? It is my opinion that the ideal projection is still the original Stenvers view. This gives the anatomic landmarks undistorted, in the clearest and most logical fashion and gives a survey over some of the possible routes of propagation of the infection.

The Stenvers technic has two disadvantages: (1) We need separate pictures for each side. (2) While the examination can be well made on a cooperative adult, on very young and excited children it is very difficult to get a set of two well comparable views.

The base-view has been given preference in many institutions since the epochal publications of Taylor. As much as we are indebted to Taylor for his great contribution in making us "petrositis-conscious," I feel that an honest discussion as to whether the Taylor view is actually an ideal projection for a key plate is now justified. This question has been raised in recent publications and discussions. The posture with the maximum posterior bending of the skull is most inconvenient for the patient, and on individuals with a short neck the results are unsatisfactory. The Taylor view has the advantage that both petrosae are shown undistorted and on one exposure. The anatomic landmarks, however, are not as clear and well defined as on the Stenvers view, important structures are superimposed upon each other and the important upper pyramidal ridge is not visualized at all. The varying extent of the pharyngeal air space may occasionally overlap and simulate a decalcification of one petrous apex.

I wish to present two other views which I consider very useful for key-plates and which have to a certain degree been neglected. One is known as the "exaggerated base view." The roentgenologic technic is simple, both pyramids are visualized in one film, moderately distorted, but giving a clear visualization of the upper petrous border down to the tip. While anatomic relations and structural details are not as well presented as on the Stenvers view, I feel that it still has value where the Taylor view was not satisfactory.

The other technic I wish to recommend as particularly useful in children is based on the old principle of projecting the petrous pyramids into the orbital spaces, and was first used by Schueller. This view, I feel, should not have been discarded in favor of the Taylor view. Its technic is very simple and agreeable to the patient who is placed comfortably on his back; we obtain routinely good films even on very young and very unruly children. The anteroposterior direction is used because the natural posture with the face up makes children unapprehensive and easy to manage. Moreover, according to the laws of rotentgenologic projection this will enlarge the orbits in relation to the pyramids which is further aided in shortening the target-film distance to 25 to 28 inches.

The view demonstrates both petrosae on one film, with the clear outlining of the upper pyramidal ridges, perilabyrintheal cells, inner auricular canals and semicircular ducts. The only disadvantages are the moderate degree of fore-shortening and the fact that the innermost portion of the tip is occasionally not visualized without superimposition. The picture, however, is unobstructed to a point medial from the internal auricular canal. Upon views taken for comparison in the Taylor and in the just mentioned anteroposterior intraorbital projection, the latter gave distinctly better details and clearer anatomic landmarks.

Summarizing, I would like to state that in institutions where for thorough and conscientious study of the petrositis problem key-plates are routinely taken, the projections developed from Stenvers technic (Stenvers, Arcelin, Loew-Beer, and others) should be made whenever possible. These films will give information superior to all other projections particularly when stereoscopic technic is used.

In young children nad patients who are unable to cooperate, a view projecting the petrous pyramids into the orbital cavities will give more information as to

anatomic landmarks and bone details than the Taylor view of the base which is commonly used.

When true petrositis has developed, all different projections should be used, combined, will give the best possible information.

Dr. H. L. Ford, Champaign, Illinois: It seems to me that both these papers are excellent presentations. Dr. Glatt's paper dealt at length with histologic pathologic changes in the mastoid structure, and I think he brought out further the probable changes occurring in the schlerotic type of mastoid with its increased blood supply of the epitympanum and the large celled pneumatic type with its poorer blood supply.

His case report is very interesting, as an example of the much dreaded "mucosus otitis." The patient experiences a sticky pain in the ear, a scanty discharge follows myringotomy or spontaneous perforation, but the *hearing* stays markedly depressed. The patient may go for several months conscious only of an "organ feeling" and poor hearing. Such a history, and especially cultures *early*, should lead us to study all such cases with the x-ray, as with the pictures we can spot destruction early and avoid the fatal complications which pneumococcus type three is prone to produce. They are asymptomatic—much like the diabetic cases.

One other point relative to catheter inflation: Kopetsky differentiates between facultative and obligatory aerobes—streptococcus pyogenes being an example of the latter. It may therefore be true, that in some cases, ventilation serves a definite purpose.

Dr. Morwitz's paper on roentgenographic aspects of petrositis, I thought was very good. I am wondering why, however, he did not mention the frontal view—the projection of the petrosa into the orbits. It is much easier to take. There is less superimposition of structures, and it reveals the *superior* petrosal margin more clearly and this is where probably the greater number of perforations take place. I have followed the technique of McMillan, of the Massachusetts Ear Infirmary—postero-anterior with nose and forehead down—plate horizontal and another anteroposterior, a reverse frontal position with chin in and plate elevated 23 degrees. This latter position is used especially for children, but throws the petrosa higher.

The otologist owes it to the patient and to himself to familiarize himself with the x-ray findings. Limitations of petrosal films should be realized, and venous spaces should not be confused with pneumatic spaces. It is important, as Dr. Morwitz has mentioned, to take an initial "key" picture and then subsequent and repeated pictures and views, using the same *technical* factors. I feel you cannot get too many pictures if you are in doubt in a particular case.

Dr. Joseph C. Beck, Chicago: I have talked so much in my day that I thought I would escape this time, but now that they have honored me by calling upon me, I am glad to pay compliments to the authors and discussers who have covered this subject so well. I listened to see if I could pick up some points to discuss, but they covered the subject so well that there is really very little to say. I would like to emphasize the functional tests. That is what the ear is for, namely, hearing, although some people seem to feel that they are to be pinned back. They are also for hearing and not for mastoid operations which have come into vogue since operators have become so efficient. Many are operated upon when the indications are not 100 per cent. However, the hearing is the important thing. Dr. Glatt emphasized the importance of tests in the adult. That is one thing but in children it is more difficult. Dr. Morwitz did not dwell upon some of the roentgenologic demonstrations. We, at the Mount Sinai Hospital, feel that we are very fortunate in having coworkers who will sit down with us and compare the changes that are going on in these mastoids and petrosae, and that is an important factor which many men in smaller cities do not have an opportunity of doing. It is really wonderful what an x-ray man can read into the picture by his experience and opportunity to study the plates.

Nothing was mentioned about drugs, that is, that drugs are or may be a masker of symptoms. In this past season of mastoid flurries I have seen cases in my own practice and in consultation where that has taken place. The doctor congratulates himself upon the fact that the child is fine and an operation has been avoided, but when you test the hearing you will find it is bad—the drum looked alright, but suddenly the patient developed meningitis. The value of this wonderful drug sulfanilamide has, as yet, not been definitely proven. How good or how bad it is we do not know, but I have seen these results and I want to state them as facts.

The gentlemen who opened the discussions are all of the right age in otology, and they brought out some excellent points—one truth in particular, that there is an individual to be considered in each case. We cannot make any uniformity of rule, but we can go away from this place very much benefited by this discussion, and I am pleased to have been here.

Dr. M. A. Glatt, Chicago (closing): I wish to thank all the discussers. I thought, at first, that this was rather an elementary subject and presented it with some hesitancy, but I now realize that some of the old timers feel that there are still many points needing clarification and discussion. I want to emphasize that all my patients, whether in private practice or those on my service at the Illinois Eye and Ear Infirmary, are always studied from an angle of any possible existing or of a latent form complication and more so when a simple or a radical mastoid operation is contemplated. While the patient, whose history I have cited, was in the clinic for possibly 15 minutes, sufficient studies were carried out by my associates to make a tentative diagnosis of a serious lesion but, regrettingly, they were not alert enough to immediately hospitalize him.

Some of you may still recollect the old time treatment of an ice-bag to an infected ear. A number of those patients developed surgical mastoiditis and the otolaryngologist was blamed for faulty treatment. Nevertheless, there are some who still adhere to this practice. With reference to Dr. Musgrave's remarks, I wish to state that most of these patients, whether

children or adults, are under the care of either the general practitioner or pediatrician and receive the usual care of the nasopharynx and it is to them that this discussion is directed. It is true that a number with otitis media recover without incising of the ear drum and, I am sure, there are many instances where inflation has been beneficial. On the other hand, there are numerous instances where a surgical mastoiditis developed after inflation. Here again an explanation must be given to the patient that the treatment was not the factor in producing it. It is therefore, im- our difficulty—proper interpretation of films as to whether it is a surgical petrositis or a medical petro- portant to be cautious with this method of treatment. There are many other claims by advocates for certain methods of treatments, but when all facts are analysed it is evident that there are many variabilities, to some of which I have called attention in this paper that have a bearing on the final result. I may again re- peat that it is important to bear in mind that besides the disappearance of all symptoms, an ear infection cannot be considered cured and out of danger when the function has not returned to its former level.

Dr. S. M. Morwitz, Chicago (closing): The main objective of my paper was to emphasize the importance of cooperation between the otologist and the roentgenol- ogist. Although actual surgical petrosities cases are not common, we are often confronted with the question of whether it is in the mastoid or labyrinth or petrous process, and I think that only by proper cooperation can we get more experience in interpreting films of these regions. Rotentgenographically, I think that is sitis.

INTERDEPENDENCE OF A PUBLIC CHILD HEALTH PROGRAM TO THE PRACTICE OF OBSTETRICS AND PEDIATRICS

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Since health programs flourish only as the result of coordinated effort, we appreciate the opportunity to discuss this subject before this group. Many of you have given evidence of a sincere desire to bring about a better understanding and better cooperation between the Health Department and organized medicine and the educational authorities.

A few years ago we talked about the interest we had in the health and life of our children—today the word interest has changed to obligation—so the obligation we have to promote the health and welfare of children is discussed. The obligation has been impelled by many considerations.

1. Excessive maternal and neonatal mortality.
2. Records of the high percentage of remedial physical defects existing among children of all ages.
3. High mortality and morbidity rate from communicable diseases, many of them preventable.
4. Low vaccinal and immunizational status in diphtheria, smallpox, scarlet fever, whooping cough, typhoid fever, etc.
5. Retardation and absenteeism in the schools resulting from illness much of it unnecessary.
6. Failure of the health habit era to make the child desire health.
7. Bodily inefficiency as a result of poor nutrition—faulty food habits, overactivity, insufficient amount of rest.
8. Slow decrease in defects of the mouth and teeth.
9. Tuberculosis statistics, especially here in Illinois where there are no State sanatoria and the policy has been to localize almost entirely the burden of official anti-tuberculosis work. Since 1932 the annual mortality rate from tuberculosis has declined 23.5% in Massachusetts; 15.4% in Pennsylvania; 11.4% in New York; and 8.3% in Illinois.

These and many other conditions that might be mentioned caused an intelligent society to reenforce childhood life and set it in new currents. We are a people who glory in a standard of culture equal to the strain of a diversity of opinion on many subjects but here is a subject in which we are a unit.

The obligation is further impelled by the custodial situation in our state. On April 1 this year there was the following child and juvenile population in the custodial institutions of Illinois.

	1939	1920
Lincoln State School and Colony	4,051	2,024
Dixon State Hospital	3,672	299
Illinois School for the Blind	237	215
Illinois School for the Deaf	529	359
Illinois Eye and Ear Infirmary	94	115
Illinois Soldiers' and Sailors Children's School	616	333
St. Charles School for Boys	623	851
State Training Schools for Girls	316	449

At Lincoln, Dixon and the Eye and Ear Infirmary there are both children and adults. There are some students at both the schools for the deaf and the blind beyond the age of twenty-one

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years. But what does this population represent? It is first of all an effort to discharge the obligation of the state to provide living conditions for children who cannot be cared for adequately in any home because of the peculiar nature of their maladies. It represents also the effort of the state to salvage as many as possible to some degree of help and of responsible citizenship. Furthermore, it is a series of laboratories, sociological and medical, in which may be worked out at no risk or cost to these children and only for their benefit some of the problems of care and improvement of such cases, and lastly, the most responsible and efficient custodians of these institutions will heartily agree that they represent in part the failure of society to prevent much of the dependency and delinquency found here, which in turn frequently bears a relation to poor physical conditions.

Go over the etiology of the maladies of many of these children and you have a mixed sociologic, psychopathic and histologic pathology. But all too frequently comes out of it the horrid results of the loathsome venereal diseases, of malnutrition, of trachoma, of birth palsy, of psychopathic heredity, of tuberculosis, and undiscovered or uncorrected physical defects. This brief mention includes predominantly causes which are preventable.

In order to assist in solving the problems as well as to extend and improve the maternal and child health services in Illinois, the State Department of Public Health during the past 20 years, especially through the personnel of its Division of Child Hygiene and Public Health Nursing (physicians, nurses, nutritionists), has carried out an official child health program, an outline of which is here presented.

Away back when

1. We had the Better Babies Conference which though somewhat crude and very general in scope, nevertheless provided a nucleus for some phases of the present set-up. In self-defense fond parents consulted the family physician about little Katy's anthropologic irregularities.

2. The Crippled Children's Clinic where we saw not only deformities of the body but defects of vision, hearing, etc., as well as problems in nutrition and mental hygiene.

District Supervising Nurses

About this time the state was divided into districts with a qualified public health nurse supervising a number of counties. The function of each district supervising nurse is to know the health needs of each community within her district; to confer with the local physicians, dentists, school authorities and other leaders relative to these needs, and plan suitable programs with them. They are active in promoting and assisting in immunization programs with emphasis on the importance of protecting the infant and preschool child. Through the schools the nurses promote new nursing service, assist doctors with medical examination of children and summer round-up programs. They plan for the general promotion and extension of the Maternity and Child Hygiene Nursing Service. This planning and work on the part of the supervising nurses creates a demand for the services of other members of the staff.

3. *Infant Mortality Surveys*

One which I made in 1925 in nine counties of the state mostly in Southern Illinois with a mortality rate of over 100 for infants under one year of age. The two outstanding causes of death as elicited from death certificates and interviews with physicians were found to be syphilis and prematurity.

4. *Breast Feeding Demonstrations*

In this connection we quote from a report by one of the local physicians: "A Breast Feeding Demonstration may be made a valuable public health work from the educational standpoint and is instructive both to the medical profession and the community at large."

5. *Organization* of Mothers' Study Clubs covering prenatal and infant care.

6. *Talks* on preventive medicine and demonstrations to civic organizations and educational groups.

7. *Stimulation* of interest in hygiene of the preschool child.

8. *Educational* campaigns on diphtheria and typhoid immunization and smallpox vaccination.

9. *Child Health Day and Health Promotion Week.*

10. *Control* of milk and water supply.

11. *Maternity* and social hygiene institutes for nurses throughout the state.

12. *Schools* of health instruction for members of the Federated Women's Clubs in order to interest them in personal, family, and com-

munity health and assist in formation of suitable local programs.

13. *Medical School Inspection*

The Division personnel assisted local physicians with the physical examination of thousands of children of all ages. Parents were present to witness the examination and discuss the findings with the doctor.

14. *School Health Appraisals*

Many schools requested a personal study of their school plant; the medical and dental service program, immunization and nursing service.

Literature

Health Messenger—official organ of the Department. Most of the pamphlets, booklets, prenatal letters, etc., were and are made up of manuscripts written or approved by obstetricians and pediatricians in the state.

Films, slides, posters and the radio all played a part in this stupendous effort to make the public health movement felt and heard throughout Illinois.

But Time Marches On

In February, 1936, funds for Maternal and Child Health Services became available under the Social Security Act.

The responsibility for developing and submitting a plan of activities devolved upon the State Department of Health where it rightfully belonged since the needs of the people in various sections of the state differ in many respects because of special geographic, racial, agricultural, industrial or educational conditions. The state has this data and should know about what type of program can be carried out successfully in the various localities and communities.

Most of this work has been planned for rural areas because of the need and also because of the fact that the Federal Act makes it clear that the grants are intended to extend and improve conditions primarily for mothers and children living in rural areas or areas of economic distress.

During the past three years the bond of relationship between the Department and the practice of obstetrics and pediatrics has been rather definitely outlined through the appointment of state advisory committees in maternal and child hygiene, and also through the recommendations of the Academy of Pediatrics. Every county in the state has a maternity hygiene committee consisting of local physicians.

The two obstetricians on the Staff of the Division of Child Hygiene have met with the County Medical Societies in the state and have addressed many lay groups. Twenty-three talks have been given so far this year by one of these obstetricians.

Postgraduate courses in obstetrics and pediatrics have been given at the University of Illinois for physicians in the state in 1937 and 1938 with an attendance of over 300. These courses are to be repeated this summer. The material presented covers the common problems encountered by the general practitioner.

During 1937, 1938, and up to the present month, 1939, obstetricians and pediatricians in the state have given 256 talks, 134 in obstetrics and 122 in pediatrics, to medical groups.

Public health nurses were urged to start staff educational programs to improve the quality of nursing service rendered to maternity and infant cases. Many organizations have developed better health teaching methods and are using community resources to better advantage.

Further Activities

Joint nursing service in operation in 21 counties. Fifty per cent. of nurse's salary and travel expense is met by the state and fifty per cent. by the county. A generalized program is carried on with special emphasis on maternity and infancy work.

Assistance to Cities

Nurses are assigned to Visiting Nurse Associations in some five or six of the cities for the extension and improvement in their maternal and child health service.

Maternity Institutes

In 1938, an able instructor from the Maternity Center Association, New York, conducted eight maternity institutes in Illinois for nurses with an attendance of 437. This large registration is most gratifying since it was voluntary and certainly demonstrates an earnest desire on the part of this group to secure a review of basic theory, skills and techniques.

Field Visits

One hundred and ninety field visits were made with nurses doing maternity and infancy nursing.

Nursing services were given to Well Baby Conferences. Where such a conference was being planned, help was given in organization. Survey suggestions were made, also how to make health examinations available for children, and the type of service to be rendered. Procedures for conducting the conferences were approved by the Medical Advisory Committee.

Delivery Service

A delivery service using private duty nurses was developed in four counties—Fulton, Greene, Hancock, Massac—under the auspices of the County Medical Society cooperating with the Division of Child Hygiene. The purpose is to give trained assistance in home deliveries to the doctor where social or medical findings indicate that nursing assistance is needed and cannot otherwise be secured. *Maternity Demonstration* service developed in five counties—Hancock, Greene, Ford, Fulton, and Vermilion. These counties were chosen because of interest on the part of the county medical group, above average maternal and infant death rate, and also because they were typical needy rural areas with many home deliveries and inadequate hospital facilities. The Medical Advisory Committee assisted the nurse in planning her program.

Cases actively registered for maternity and child health supervision:

	Cases Registered	Home Visits Made
Hancock County	598	1137
Greene County	249	718
Ford County	692	1894
Fulton County	285	660
Vermilion County	just started	...
Total in 4 counties	1824	4409

Programs have been developed especially throughout the southern part of the state for improving the care of premature babies. Several improvised incubators are in operation. All hospitals discharging premature infants are being asked to see that these babies have nursing supervision at home. Further, hospitals that lack facilities for the care of the premature infant have been encouraged to improvise and add suitable equipment when the same seems feasible.

Before leaving the subject of maternity hygiene, I should like to call your attention to

Mortality Trends.

The last thirty years witnessed remarkable progress in making childbirth safer in Illinois. The risk of puerperal septicemia, or childbed fever, for example, is now but a fraction of what it was three decades ago. From a rate of 8.9 in 1918 to 3.3 in 1938, or about one-third fewer mothers dying during the year just past, shows marked improvement.

Infant mortality decline is most significant when we consider that the rate in 1918 was 102.4 and in 1938 was 41. Further, the rate in 1934 was 52.2, so in four years we have saved the lives of nearly 1,000 infants.

The improvement in the infant and maternal death rate is undoubtedly due in no small measure to the expansion and intensification of public health services. There can be no doubt that programs in maternity and child hygiene have been responsible for the improvement where progress has been observed.

In this same period the birth rate dropped from 20.1 to 14.6 in 1937 and went up to 15.4 in 1938 which is the highest rate since 1931. (See graphs and charts in Illinois Department of Public Health Exhibit).

Immunization Program

Also in Southern Illinois nurses assisted local doctors with about 8,000 typhoid immunizations, 3,000 vaccinations for smallpox, 4,000 diphtheria immunizations, more than 1,000 tuberculin tests and about 1800 Schick Tests.

Social Hygiene and Parent Education Program is now well established as an integral part of the services of the Division of Child Hygiene and Public Health Nursing. The program for the year was planned with four objectives in mind:

1. Continuing services to significant special groups and communities already reached in an earlier program.

2. Initial programs in new communities with particular emphasis on small rural groups in the area affected by the flood of 1937.

3. Continued development of adult education through service to parents, teachers and civic groups to the end that local communities be encouraged to promote a continuing community social hygiene program.

4. Stimulation of public opinion in this field through individual and group study made possible by the availability of more adequate library service.

Syphilis Control Program

In connection with the work in social hygiene, may I refer to the syphilis control program for a small city which is in effect at the present time in the Champaign-Urbana Health District. Briefly outlined, the plan approved by the State Department of Public Health and the local physicians makes it possible:

1. For all cases of infectious syphilis to be treated in the private office of physicians selected by the patient.
2. The physicians are paid for all treatments, either the regular fee by the patient, or they receive from local funds payment at the rate agreed upon.
3. A complete follow-up system is established for all cases.
4. A defined minimum standard of treatment is established for all cases which it would seem should effectively control infectious syphilis, as well as prevent the development of late syphilitic manifestation in the great majority of cases.
5. A committee of representative physicians is established as the offcial control unit of the Board of Health in this district.

Nutrition

During 1937 one nutritionist was assigned to Southern Illinois. Her program included individual family consultations as requested bp the doctors and the nurses, lectures to lay groups, talks in schools and work coordinated with Home and Farm Bureaus.

For the most part, the plan of work for the other nutritionist consisted of consultant service to staff nurses, schools, school cafeteria and lunch-room managers.

Talks on nutrition were given to groups of mothers, teachers, nurses and school pupils.

The work of the nutritionists makes us realize that the nutritional defects of children are due not so much in our state to a lack of food but to an unbalanced diet. "What to eat" is more than a cook's question as she considers the palates and appetites of her family. "What to eat" is a question to be answered for millions of children who without its answer must suffer some form of deprivation disease not measurable merely in terms pathological and physiological but in terms of efficiency and happiness. And this is a problem which can be met with an adequate expenditure in education in schools, in homes, in doctors' offices and health centers.

Conclusion

Throughout the years the Division of Child Hygiene and Public Health Nursing has had the finest cooperation from organized medicine, from County Medical Societies and from doctors everywhere throughout the state.

The important policies and programs have been worked out with the Educational Committee of the Illinois State Medical Society.

For years the medical staff of the Division have given very few inoculations for diphtheria or smallpox, preferring to leave this work for local physicians.

In the new Federal program the Department has met with the cooperation of the Academy of Pediatrics, the County Medical Societies and the obstetricians.

Unfortunately, there are those who think of the physicians in the Department as purveyors of State Medicine. This is most unjust as there is not a group of people anywhere who are in a position to so fully appreciate what the family physician is doing and can do for the people in his community.

Let us continue to work together. We have help for you and you have help for us. What concerns us concerns you and what concerns you concerns us. Let us know your needs according to our special services and help us to fulfill our functions so that there may live in the soul of the state the words of Mrs. Browning:

"Do you hear the children weeping, O my brothers,
Ere the sorrow comes with years."
For we know
"That the child's sob in the silence curses deeper
Than the strong man in his wrath."

DISCUSSION

Dr. Charles Newberger, Chicago: The facts brought out in Dr. Ball's paper, so far as puerperal sepsis is concerned, leads me to bring this to your attention: Statistics show that in 1928 the maternal death rate in Chicago was 56 in 10,000; last year there were 27 in 10,000. Undoubtedly it is the result of such work as done by the Department of Child Hygiene and the Maternal Welfare Committees and the harder work of obstetrical staffs in the hospitals that has brought this about.

Dr. G. F. Munns, Winnetka: I think we should be grateful to Dr. Ball for this very good description of the activities of this department. I have had occasion

to work with them this year and know how cooperative and fine they are in every way in helping us put across our project, and we appreciate it a great deal. I think that the work in the State Department will keep on increasing during the next years. As their function seems to be chiefly educational it is something we can back to our fullest energy.

I wish to thank Dr. Ball again for letting us see in a very comprehensive way all the activities that take place in the state for the care of children and mothers.

Dr. Gerald Cline, Bloomington: Downstate we all agree that our close cooperation with the Health Department has been a great help to us. Personally it has been a great privilege to have such people back of us.

Dr. Clifford Grulee, Evanston: I have worked on this problem for many years and the thing that has struck me has been the absolute cooperation between the Health Department, the State Medical Society and the special groups. I must confess that in the early years I was very surprised at this because I had heard so much about friction. So far as I can see there has been very little friction. I think, though, that many of us who have been at this work a long time realize better than others who take it up now that it was a long pull. It is not a question of getting something done so much as a question of getting people to know that it should be done and how it should be done. In other words, if we were a totalitarian state it would be possible to choke down all the thoughts and ideas we have had which under our ways of life I think would be a grave mistake. I think we should go ahead as we have, and that is in the gradual accumulation of our own knowledge and carrying this over to the people in the state. There is no question in my mind that the ultimate solution of public health is the private physician; I do not see how it can be otherwise, and I very much hope that the private physician will take this into his own hands as he has the possibility of doing and not allow the government to do it. That is how all the public health people that I have talked to feel, from those in Washington on down, and I have talked to most of them. We feel that the thing lies in the hands of the practicing physician and it is up to him to take the plow and break the furrow.

PREGNANCY IN DOUBLE UTERUS

MAURICE P. ROGERS, M. D.

AND

BERGET H. BLOCKSOM, JR., M. D.

ROCKFORD, ILLINOIS

The entire question of the etiology of congenital malformations has long been an intriguing subject for the biologist, and various causes have been ascribed for the occurrence of congenital defects. Why the germ plasm should fail to develop in such a manner as to permit failures of fusion such as cleft palate, spina bifida, or double uterus, has yet to be definitely proven, although two general hypotheses might be found tenable:

1. Inherent defects in the germ plasm.
2. Unfavorable environment.

Unquestionably some ova are inadequate at the time of fertilization, as is shown by the studies of Huber and Kellicott, and anomalies may have their origin in the primary division of the ovum. The development of a grossly imperfect ovum is fortunately terminated by the failure of the fetus to develop to maturity, with a resultant miscarriage or premature delivery of the stillborn, malformed fetus.

Stockard and Hertwig have shown that slight variation in the chemical constituents of the medium surrounding fish embryos, leads to the production of cyclopia in half of the embryos, provided the change in environment was brought about immediately after fertilization, while if this change were delayed for fifteen hours, the eyes developed normally.

The uterus, fallopian tubes and upper portion of the vagina develop from the Müllerian ducts. The first evidence of the appearance of these ducts is seen in embryos ten millimeters in length, approximately one month old, and they first consist of a thickening of the epithelium of the urogenital fold. They first make their appearance in the third thoracic segment, and migrate and lengthen in their descent toward the pelvis. Shortly a lumen appears in each of the Müllerian bodies, with a fusion which begins at the caudal extremity to form the uterovaginal canal. The Müllerian bodies are originally apposed by their outer surfaces, resulting in a double canal. An absorption of the septum between the two cavities then normally occurs. From this point on, a rapid differentiation in sex is apparent. In the female the fused Müllerian bodies continue to develop and migrate, until the uterovaginal canal assumes its normal relationship. A failure of the Müllerian bodies to contact each other during the development of the fetus, results in almost a complete loss of both, usually with a failure to find in the fetus even a rudimentary uterine body. If this is regarded as one extreme, the slight dimpling or notching which is occasionally seen in the middle of the uterine fundus probably represents the other. In between these two lie a large number of interesting anomalies with which the practitioner is often called upon to deal. Anomalies

Fig. 1. Double uterus, demonstrated by injection of iodized oil in uterine cavites. (Author's Case)

in fusion of the Müllerian bodies are frequently associated with improper or incomplete migration of the hindgut, resulting in rectovaginal fistula, or vulvovaginal anus. It is our wish to report such a case.

CASE REPORT

Mrs. L. W., aged twenty-one, presented herself for examination on April 12, 1938. Her family history was noncontributory. Her present history revealed that she had been married on December 25, 1937, and that her last menstrual period was in January, 1938. She complained of shortness of breath and a consciousness of abnormal cardiac activity, but said she had never had dependent edema, orthopnea, or other evidence of acute cardiac decompensation. She stated that she had been born with an imperforate anus, two vaginas, and two wombs, and that at eighteen months of age some type of operation had been performed for relief of the condition. She had been markedly constipated all her life, and had taken three cascara tablets or their equivalent nightly, often without results. She had at times gone as long as two weeks without a bowel movement. Her vision had always been weak, and her eyes were correctable only to 20/70. She was subject to recurrent attacks of tonsillitis. Her urinary history was negative and she had never had rheumatism, chorea or scarlet fever.

Physical examination revealed a rather poorly developed, undernourished white female. Her pupils were equal, regular, and reacted to light and accommodation; her ocular fundi were normal; ears, throat, cervical glands and lungs were negative; the left heart border was well within the mammary line, and on auscultation a blowing, presystolic murmur was heard over the pulmonic area and transmitted over the entire chest. Examination of the abdomen revealed no masses or areas of rigidity. In the perineum there was a slight dimpling of the cutaneous tissue at the normal site of the anus. The external genitalia appeared normal. One centimeter inside the vaginal orifice there was an opening one and one-half centimeters in diameter, which proved to be the rectal opening, the mucosa of which was continuous with that of the vagina. Slightly higher in the vagina a firm septum was encountered, running perpendicularly from the anterior to the posterior wall, and terminating in normal appearing fornicies. Located in either vault was a cervix with a short portio vaginalis, each with an apparent normal os. Bimanual examination revealed a double uterus, both of which were enlarged, the right being approximately four times normal size.

A diagnosis of pregnancy in a didelphic uterus, rectovaginal fistula, and congenital heart disease was made. The course of her pregnancy was not remarkable, except for the development of a low grade secondary anemia and a rather marked hypotension, her systolic pressure going down to seventy, with a diastolic of forty. An x-ray which was taken September 6, 1938, demonstrated a fetus of approximately seven and one-half months, lying entirely in the right side of the abdomen, with a vertex presentation. On October 23, 1938, the patient began to experience intermittent abdominal pain and there was a slight vaginal discharge. Because of her cardiac condition, abdominal delivery was decided upon. The abdomen was opened and the presence of a double uterus confirmed. The left fundus was approximaely three times the size of a normal uterus. The corpus luteum was discovered in the right ovary. A full term, living male child was delivered by the classical method. Pituitrin introduced into the uterus produced firm contraction of the gravid fundus, but had no apparent affect on the non-pregnant side. The patient made an uneventful recovery. Examination of the baby failed to elicit the presence of any congenital defect.

On the occasion of her final postpartum examination, iodized oil was introduced by cannula into both uterine fundi, and x-ray studies were made of the relationship of the two uterine cavities.

Reconstruction of the rectal canal, with a closure of the rectovaginal fistula is contemplated at a future date.

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PSYCHOSES IN CHILDREN

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The Institute for Juvenile Research examines by appointment some 1,000 children a year at

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the headquarters clinic, in addition to the many hundreds seen at the Juvenile Court branch and the various community branch clinics throughout the state. There pass through its diagnostic and admissions service, however, many cases, emergency and otherwise, which are of necessity rejected or referred to other agencies and clinics after the presenting problem has been thoroughly evaluated. In this latter group of cases are to be found many of the young psychotics with whom this paper is concerned, and I am presenting a review of this material as it has passed through the admissions service from January 1, 1938, to January 1, 1939.

Psychotic children represent a very small percentage of our state hospital population and this is especially true of the prepubescent child. At the Cook County Psychopathic Hospital which serves as the admission center for the entire metropolitan district of Chicago and Cook County, Dr. Gerty's statistics for this same year, 1938, indicate that only twenty children under fifteen years of age were admitted, ten female, and ten male. Of the ten males two were juvenile paretics, one a case of organic brain disease, undifferentiated, one, central nervous system lues, other than paresis, one mental defective with, and one without psychosis, and four schizophrenics. Of the ten female children one was a juvenile paretic, one a case of cerebral neoplasm with psychosis, one a mental defective with psychosis, one undiagnosed psychosis, one without psychosis, and five schizophrenics. All told, there were 6,182 admissions to the hospital with 1,152 schizophrenic cases as first admissions and 559 schizophrenic readmissions, a total of 1,711.

Kasanin and Kaufman[1] reported that out of a total of 6,000 patients admitted to the Boston Psychopathic Hospital between 1923 and 1925, 160 were under 16 years; 65 of these were psychotic, including 21 schizophrenics. The one-year difference in age level may account for the larger figures in the Boston group.

It has long been known that the incidence of psychosis increases rapidly after puberty, a period of important psychobiological changes, when children predisposed to schizophrenia become exposed to traumatic environmental factors of a sufficiently severe degree to precipitate a psychosis upon an unstable, poorly integrated personality structure.

CASE MATERIAL

During the last year our psychotic children have included one case of juvenile paresis in a colored boy of twelve, and one of juvenile Huntington's chorea in a girl of fifteen. Both of these children were immediately committed to state hospitals through the Psychopathic Hospital. Three girls from training and correctional schools, with periods of severe excitement were seen. They were of that variety of case seen almost exclusively in girls, which often requires hospitalization and which, for lack of a better diagnosis, is usually classified as "psychopathic personality with periods of excitement." These girls, all of them extremely interesting neurotic character and personality studies, often act out in an impulsive fashion during violent rages and excited states and frequently require constant institutional care.

Two cases of mental deficiency were seen in which similar periods of excitement, sufficiently severe to require physical restraint, were part of the general picture. One case of so-called idiopathic epilepsy with suggestive clouded states was also examined.

There were five cases, which we have added to a special group under separate study, of preadolescent children who have carried out serious suicidal attempts, and who, in many cases, at the time of the active suicidal drive at least, might well have been termed psychotic. One of these cases, a boy of 12, seen a year and a half after he had shot himself through the frontal and temporal lobes of the brain, had developed evidence of a definite organic psychosis associated with the actual cerebral damage, and commitment was advised.

Two depressive reactions unassociated with actual suicidal activities were not deemed sufficiently serious to warrant the consideration of institutional care.

All varieties of mental deficiency have been observed including some with physical stigmata, pronounced organic brain lesions, and focal neurological findings, secondary to birth injury, encephalitis, meningitis, etc. There is also the so-called cerebral agenesis variety without focal symptoms, and a type, also without neurological findings, probably due to trauma. These pure defectives are mentioned here because they definitely differ from the peculiar children in the

constitutional group of schizophrenics that I shall describe, although the latter often appear on the surface to be mentally deficient.

The so-called postencephalitic group of cases is one of the most difficult to differentiate from the birth-trauma varieties. These cases are often like those described by Jasper[7] and his associates, and like many of Sherman's[9] cases, but whenever in our series a schizophrenic picture has complicated the case I have classified it with the schizophrenic reaction group. The serious behavior disorders occasionally associated with pronounced sexual maladjustment are usually characterized by hyperactivity, poor school progress, distractibility, inability to associate and get along with other children, occasionally mild motor release or epileptic phenomena, and rarely definite choreatic or choreo-athetotic syndromes. In only one case, a true Parkinsonian state occurred. The importance of these cases cannot be overemphasized because they affect prepubescent children, and present a dubious prognosis. Many of these children are overprotected and make only slow progress as they gradually come under the influence of socializing forces.

We are now left with the schizophrenic reaction group regarding which there is so much controversy, frequently even so far as diagnosis is concerned. Twenty such cases were seen during 1938.

For purposes of presentation I have divided the schizophrenic reaction group into three subgroups, although it will obviously be difficult to categorize many of the cases as finely as such a division would imply. As it is almost impossible to obtain all of the anamnestic details that are so necessary in order to secure an accurate impression of personality characteristics and their changes, I may be justly subject to criticism when I say that the children in the first two groups appeared to have made a fairly adequate adjustment until shortly before the onset of psychotic symptoms. Nevertheless there does seem to be a definite difference which I shall later describe, between the cases mentioned in the postpubescent and prepubescent groups, and the constitutional group. The first is considered separately from the second mainly because it consists of cases in which psychotic symptoms appeared after puberty.

POSTPUBESCENT GROUP

This group comprising four of the 20 cases, consists of children all of whom had made a fairly satisfactory adjustment on the surface, at least, until shortly before the onset of active symptoms. These cases resemble most closely the usual late adolescent or early adulthood type of schizophrenic psychosis presenting what is usually accepted as the typical pre-psychotic schizoid personality and fairly clear-cut anamnestic accounts of psychogenic precipitating factors. The following case exemplifies this group of cases:

CASE REPORTS

1. A 16-year-old boy was seen on June 14, 1938, when the family physician told the parents that the boy seemed to be a mental case, and advised interesting the patient in a hobby of some sort.

A maternal uncle is said to be a mental case.

The history is that of a sensitive youngster, the second of three male children. He had always been markedly attached to his mother and quite dependent upon her. While described as a "good and obedient child," he had never had many friends, had always been stubborn and unhappy, and had found it difficult to show affection to his parents or siblings.

Though he seemed to have made a satisfactory adjustment up until the time he entered high school at 12, a definite change was soon noticed. He began to truant without his parents' knowledge, soon lost all interest in his studies, and began to indulge in mild forms of anti-social behavior in the company of other boys. Six months before he was seen at the Institute, he was brought home in a patrol wagon and threatened with commitment to the Parental School for continued truancy.

Demonstrable psychotic symptoms appeared about four months later. He "raved" and shouted, threatened suicide, and was obsessed for a time with the idea that a friend of the family, a mortician, was putting embalming fluid into his mother's coffee. He then began to talk of sticking a knife into his mother, cutting her up, and of killing her in other ways. On occasions he threatened to "knock her brains out" if he found her crying, but whenever she actually cried he became remorseful and promised to be good and obedient. Soon he began to complain of voices in his head, and he felt he had a disease which was making him "rot away." He began to worry about dirt and dirty things, developed a handwashing compulsion, and stated that he must get away from all people, preferably to a small cottage in the North woods, where he could live alone or "rot away in peace."

He complained of the dirtiness of everything about him and wanted to join the Navy or a C. C. C. camp. He refused to get up on the morning of his appointment at the Institute insisting he would prefer to remain in bed and "rot" there.

The mother could not understand why she was such a disturbing factor to the boy and why he directed all of his resentment, antagonism, and other emotional manifestations towards her particularly since he had been her favorite among her three boys, and she had always indicated this preference to him. The father, on the other hand, felt quite guilty because he had not been sufficiently close to the boy in their earlier relationships.

The examination revealed a tall, thin, asthenic youth who presented an adolescent acne, was markedly introspective, severely hypochondriacal, withdrawn, spontaneity-lacking, and, on the surface at least, somewhat depressed. Occasionally his speech became rambling and even mildly incoherent in character and his affect was shallow and rigid. He exhibited bizarre psychosomatic delusions, and stated that his strength was slowly being drained from his body through masturbation and nocturnal seminal emissions. He asserted that he felt the fluids being drawn downward from the region of his head. There were clicking sensations in his head occurring rhythmically with each step. Suggestive hallucinatory experiences were present and described in terms of his conscience. Insight was entirely lacking and the boy reiterated time and again that he merely needed a tonic and a C. C. C. camp to be cured.

A psychometric examination revealed him to be of at least average intelligence.

He was referred immediately to the Psychopathic Hospital to enable him to have shock therapy as soon as it could be obtained for him, although it was felt that the prognosis was a very dubious one.

Since this paper is written primarily from a descriptive standpoint, I shall not attempt to speculate upon the probable dynamic factors responsible for the loss of the reality testing power of the ego, and for the appearance of schizophrenic thinking and patterns of behavior. The case cited is replete with material to suggest a strong underlying conflict in the boy over his concept of sexuality and his relationship with his mother and, of course, his father.

2. In a 14-year-old Negro girl the active psychotic symptoms were preceded by self-depreciatory statements and expressions of guilt over several so-called "sinful sexual experiences," regarding which she refused to elaborate.

3. In a 14-year-old white boy there was evidence of marked oedipus guilt expressed in his relationship to his mother who was separated from his father, and with whom he was living. There was also a pronounced hostility which he felt towards a younger brother and which he repressed only with great difficulty. This boy's catatonic episode responded favorably to metrazol therapy administered at the Chicago State Hospital, and he is now in a fairly good state of remission, almost a year following his discharge.

4. A 16-year-old white girl is a definite hebephrenic, now at the Elgin State Hospital, after having been diagnosed as non-psychotic only a year before by a competent psychiatrist. Her background is exceedingly traumatic, the family a disorganized and broken one, and she had spent many years in an institutional existence to which she had adjusted very poorly.

PREPUBESCENT GROUP

This group of cases consists of prepubescent schizophrenic reactions in children presenting no history of serious personality or constitutional difficulties preceding the onset of the psychotic symptoms. Four of the cases fit this category, and all are briefly summarized here. The last of these resembles in many respects some of the cases of the first group and is included mainly because the boy's active psychotic symptoms developed before he had begun to manifest any of the usual signs of puberty. While the anamnesis is quite similar to that of the 14-year-old catatonic mentioned earlier, who had received metrazol therapy, it is quite apparent that the outlook is more dubious for the younger boy.

CASE REPORTS

1. A 7-year-old colored girl was seen on November 18, 1938, having been referred through the Chicago Relief Administration, after she had begun to act queerly.

The family history indicates that the mother died in a state hospital in the east six months after the child was born, and shortly after her husband had deserted both of them. The child was brought up by an aunt, and has never been told about her true parents.

In August, 1938, the uncle (foster father) was committed to the Manteno State Hospital with a diagnosis of chronic alcoholism with organic changes, epilepsy, and deterioration. He had deserted frequently prior to that time. There was considerable evidence that the child had been exposed to unwholesome situations in and about the home.

In December, 1937, she was removed from school when she contracted chicken pox. She had not returned to school since that time and had had several other illnesses of a relatively minor nature. In September, 1938, a county physician was called because of the child's vague abdominal complaints and he reported that she was having hallucinatory experiences. The aunt insisted that she had noticed no peculiar behavior prior to that time, although it is obvious that mental symptoms had been present. The family was living above a garage and soon the child complained that gun-fire was occurring and that their lives were in danger. She stated that she heard men's voices saying that the aunt would be killed or injured. She sat in a corner for hours at a time and seemed depressed. She continually washed her hands and changed her clothing and expressed fear of dirt in the food. For a time she suffered from insomnia and remained awake during the night to listen to the voices. She lost weight, cried hysterically, and was afraid to be alone.

At the time she was examined she seemed to have improved considerably. The aunt stated that a great deal of improvement had been noted immediately after the family removed to quieter surroundings, from the rooms above the garage. She gave a history, too, of having been held up and struck on the head by a colored man while walking with the child two years before. The latter was extremely nervous, screaming and crying constantly for a considerable period afterward.

When the child was interviewed she stated that the voices no longer spoke to her, but in the past they had often stated that a man was coming to kill her and do things to her with a gun. Her dreams were concerned with similar sado-masochistic material, and she gave an account which indicated promiscuity in the home on the part of the aunt.

Psychological tests classified her as a borderline defective, but there was considerable scatter, and it was felt that she was probably of higher intelligence.

2. A 12-year-old Jewish girl, whose mother had been committed to a state hospital as a schizophrenic immediately following the child's birth, was seen. The mother had been divorced four years earlier. The child went to visit her in the summer of 1937 and remained until May, 1938. She was unable to get along with her mother, quarreled a great deal, and was told that her birth had been responsible for the mother's mental illness.

When she returned to Chicago she found that her father had married his housekeeper. The child became quite upset, was over-active and excited, insisted on running the household, and did the scrubbing and washing. After two weeks of this behavior she went to live with an aunt for a short time and seemed much better while away from her father and her stepmother. On her return she seemed apathetic, had to be fed, was often mute, and showed no interest in anything.

According to the history, the child had obtained an I. Q. of 142, on a psychological test performed at the school.

When she was seen at the Institute in August, 1938, she presented symptoms of indecision, negativism, blocking, mutism, dissociated affective displays and similar catatonic symptoms.

Arrangements were made for hospitalization at the Michael Reese Hospital. The patient improved, left the hospital in four weeks, is now back at school, but presents definite anxiety and compulsive symptoms.

3. An 11-year-old white boy had an attack of poliomyelitis at four. When he was nine, while the father was away from the farm on which they lived, the hired man murdered the boy's mother. The child is said to have been aware of the circumstances of his mother's death and he begged permission to be allowed to remain away from her funeral.

Shortly afterward there developed symptoms of extreme fear associated with marked temper tantrums. Later he became more and more withdrawn, soiled and wet himself, remained mute over long periods of time, experienced periods of unexplainable loud laughter, and occasionally sang in a childish fashion.

He was examined at the Institute in June of 1938 and presented fairly typical catatonic symptoms including suggestive cerea flexibilitas, mutism, and stereotyped gait.

The boy had been brought in from a rural community and since immediate hospital and sanitarium care seemed indicated, the father was so advised. There has been no follow-up study.

4. A 14-year-old Polish boy when last seen at the Institute in December, 1938, presented the picture of a typical hebephrenic, adopting hallucinatory attitndes and exhibiting facial mannerisms and sudden outbursts of silly laughter associated with a fearful, withdrawn attitude. The extremities were cold and cyanotic.

When the boy was 2½ years of age he was run over by an automobile, sustaining a fractured pelvis and leg. The fractured extremity remained shorter than the other, but in spite of this handicap the boy was anxious to become a Big League ball player. He tried to dominate other children and as a result got along poorly with them.

The parents, who are first cousins, dissolved their marriage when the boy was nine. The latter has always been resentful of his mother's second husband. He brooded over his parents' separation and during his entire twelfth year was unusually quiet and preoccupied, sitting idly and daydreaming. In June, 1937, he was graduated from the grade school at 12 with excellent grades. The following month he was admitted to the Psychopathic Hospital. He had run away from home when plans were made to have him visit his mother. He carried a toy gun with which to shoot the police. Three days before his admission to the Psychopathic Hospital, his mother visited him and then left. He had felt she was going to remain with him and his father, and when she left he gave her a disdainful look. Shortly afterward he became hyperactive, danced, and shouted, and stated that God was talking to him and directing his activities. He claimed that he was the Lord, kept the shades drawn, and the doors locked, for fear someone would harm him and shouted, "I suffer, but they're going to suffer twice as much."

The father removed him from the Manteno State Hospital against the advice of the medical staff after two months had elapsed and seemed to have no insight into the gravity of the boy's mental condition. The boy started high school in the fall of 1938, and we were asked to examine him in November. In view of the findings obtained through the psychiatric interview, mentioned briefly at the beginning of this case report, removal from school was recommended and recommitment, if it could possibly be arranged. It was found at the time of the examination that we had previously seen the boy shortly after the onset of the psychosis and had at that time advised institutional care and treatment rather than the foster home care which was desired by the father.

CONSTITUTIONAL GROUP

This group of cases differs in many respects from those just outlined. In these children we see reflected throughout the history from birth, or shortly afterwards, definite constitutional variations, at times amounting to true organic difficulties. These youngsters are often referred to as queer and different as far back as one can recall their behavior. Often they are classified as mental defectives on the basis of their response to social and school situations, and even to certain psychological tests. There may be associated constitutional inferiority, physical defects of many varieties, and occasionally even vague neurological signs.

When such children are examined it is difficult to correlate the picture they present with the subgroups mentioned earlier. There is a tendency to search for a history of trauma, encephalitis, or other possible organic etiological factors that might have produced the presenting symptoms.

Ten cases seem to fit into this group, either because the psychotic behavior per se dated back to infancy, or because there was associated mental deficiency or suggestively organic (such as epileptic) phenomena.

In some of the "organic" cases it was trauma or the influence of socializing needs that seemed responsible for the onset of the more active mental symptoms.

CASE REPORTS

1. A 15-year-old white boy, presented a history of having been peculiar and different from other children since early childhood. His mother died on the day following his birth and the boy has one sibling, a boy nine years his senior. The first three years of his life were characterized by insecurity and instability in the home situation. At six, the boy suffered a head injury, sustaining a deep laceration. By the time he was eight or nine, he was characterized as peculiar, lazy, dreamy and indifferent. He continued in high school until the time of his examination at the Institute in January, 1938, having been referred for study when he started a fire in a locker at school. He was a shy, timid, manneristic, apathetic boy, silly, incoherent in his speech productions and hallucinated. Peculiar psychosomatic delusions, a bizarre fantasy life, neologistic formations and many of the other symptoms which we ordinarily associate with hebephrenia were present. In spite of the rather severe disintegration of the normal thought processes, the boy classified as average on two psychological tests and was still attending school. The physical examination was negative. The father refused to consider commitment.

2. A 17-year-old white boy, has been seen at the Institute on various occasions since he was seven years of age. The father feels that the boy was normal until he was five when he was frightened by an explosion nearby. He began to stutter shortly afterwards. There was an old diagnosis of cerebral hemorrhage made when the child was a few months old. The developmental history is essentially normal, however.

At seven, intelligence tests classified him as a borderline defective. At 17, his intelligence quotient was ten points higher than at seven, and he was passing tests at the average adult level. There was a marked scatter and the psychologist felt the boy was not a true defective.

At 13 he was a seclusive retarded boy, called "goofy" and crazy by the other boys with whom he attempted to associate.

By the time he was 16 he had been seen by several neurologists, had been tried in various schools including a military school, and had shown little improvement.

At 17, he was a peculiar manneristic boy who presented a stuttering dysarthria and a strabismus. He had no friends, spent all of his time at home listening to the radio, and indulged in compulsive masturbation. There were suggestive auditory hallucinatory experiences, and he was careless, neglectful of his appearance and talked to himself.

3. A 14-year-old Swedish boy was seen in July, 1938. His birth and early developmental history is said to have been normal. There was considerable difficulty in school with a specific reading handicap and marked general retardation. He had frequent convulsions from the age of one year to four. There was another unusually severe seizure at eight. In May, 1938, he fell from a low-lying roof. He did not lose consciousness. He was removed to the Cook County Hospital following the development of peculiar behavior in school several days later, and a diagnosis of hysterical hemiplegia in a mentally retarded boy with a history of epilepsy, was made. The boy refused to eat or talk and had to be tube fed. He had an I. Q. of 73 and there was a previous I. Q. of 79, obtained two years before at the Montefiore School.

Thorough neurological and laboratory examinations were negative except for a peculiar left-sided hysterical paralysis.

Two weeks after his discharge he was readmitted with a diagnosis of postconvulsive state. He had experienced a severe epileptic seizure in school the day before. He was discharged once again in June.

A month later he was seen at the Institute. In the interim he had continued to act peculiarly at home. He was talkative, cried a great deal, pulled all the shades down, nailed boards on the windows, stated that people were "flashlighting" the home in order to seek him out, and heard the people on the street making slurring remarks about him. He yelled, "I'll kill them! I'll kill them!" He walked the floors wildly, had alternating periods of mutism and talkativeness, and shouted

"I've got enough sickness in my head without having chickens and dirt in my head."

At the time of his examination he was suspicious, adopted hallucinatory attitudes, and stated that the electric fan was broadcasting about him. After a time he became mute, fearful, and stubborn.

His I. Q. was 64 on the Revised Stanford-Binet, although the results were considered low because of the boy's disturbed mental condition. A careful neurological examination suggested the possibility of right-sided cerebral pathology, but the findings were extremely vague.

The boy was committed to the Elgin State Hospital, through the Psychopathic Hospital.

Two additional cases were regarded by us as relatively incipient in that there was none of the usual full-blown symptomatology which we associate with schizophrenia, and only the early withdrawal, autistic thinking, self-preoccupation, and socializing difficulties which stamped the child as different from the others in the school situation and at home.

LITERATURE

Psychoanalytic research has emphasized that the chief difference between a psychosis and a psychoneurosis consists in the fact that the latter is chiefly a conflict between the different structural parts of the mental apparatus, while in the former the relation of the mental apparatus to the external world is disturbed in a pronounced manner. With the loss of the reality testing power of the ego, there is an elaboration of the more commonly recognized symptoms of schizophrenia with which we are all familiar, many of them bizarre manifestations of instinctual tendencies. Alexander[2] feels that there may be an inherent constitutional weakness in potential schizophrenic patients, though he stresses the post-natal influences in the first two or three years of life when external forces may have been operating in the direction of making external reality unacceptable to the weak, flexible, infantile ego. While in some cases, according to Alexander, there are unusual and violent psychological influences that can explain a rejection of the environment at this stage, there is often a suggestion of other undetermined innate factors that differentiate these cases from the neurotic whose disturbances date from emotional conflicts with environmental factors at the period of the development of the conscience.

Benedek[3] has given us added psychological insight into the post-natal period. In a recent article concerned with the adaptation to reality in early infancy, she describes the process of learning in the infant, and emphasizes the importance of satisfying its needs. She speaks of the various frustations that may occur, and of the effects of these factors upon the psychic and physiologic processes of the infant whose nervous system is still immature and not completely differentiated. Disturbances in the rhythm of the child's autonomic processes are cited.

Heredity and constitution have been blamed for a great deal in contributing even directly to the production of neuropathic and psychopathic children. Yakovlev[4] has recently described congenital morphologic abnormalities of the nervous system and their functional implications. He, too, has emphasized the congenital fragility of the infant's nervous system, and the fact that reflex integration, particularly in the autonomic division, is a post-natal acquisition. He feels that idiopathic epilepsy, for example, is a "congenital functional and therefore structural malformation of a specific reflex mechanism." Singer[5] has stressed the importance of the autonomic nervous system in the etiologic and pathologic considerations of schizophrenia. He has postulated that agenesis and defects caused by lesions occurring early in life may involve the autonomic nervous system as well as the corticospinal system, to be expressed in the form of defective energy of reaction, low levels of metabolic activity, etc. Singer prefers to view the psychosis proper as the result of the action of some pathologic lesion upon a given personality, and the schizophrenic picture as a symptom complex that may result from either pathologic or "functional" changes.

Frequently the question has been raised whether it is heredity as such, in the genetic sense, or the influence of the psychotic parent that is responsible for many of the child's difficulties. Of great interest to us at the Institute has been the observation that children who were removed from a psychotic parent showed a remarkable advance in intellectual grasp as determined by the usual intelligence tests, and at the same time evidenced distinct emotional changes for the better. In two such cases intelligence quotients of 50 and 60, advanced to 90 and 100 within a period of two and three years. In one instance involving two young Jewish children, the younger child who had been in contact with

his mother during his most impressionable years, was much more seriously affected from the emotional standpoint than was his older brother who at twelve displayed typical symptoms of a well-marked compulsive character formation.

In considering the prenatal and postnatal influences, one cannot overlook the birth process as such, whether we speak of the trauma of birth in a psychological sense or of organic birth trauma. Investigative work is pointing more strongly to the importance of varying degrees of birth injury in producing neurological disorders, the etiology of which had previously been unknown. Recently Peterman[6] stated that the greatest hazard of the new-born infant is his passage through the birth canal and from 10 to 12 per cent. of all babies born in a normal delivery have some degree of damage or injury to the brain. With abnormal or pathological labors this percentage rapidly increases to nearly 100. Improvements in technique and interpretation in electroencephalography have opened a new field in the understanding of some of these vague traumatic changes. The old concept of cerebral birth palsy of the spastic or choreo-athetoid variety as the chief representative of the group of birth trauma conditions, is giving place to the feeling that trauma may occur in many parts of the brain, may vary in degree as well as location, may exert a direct influence on the child's development, and may frequently be unassociated with gross pathological changes.

We see not only mental defectives in this group of trauma cases, defectives that differ from the type associated with so-called cerebral agenesis, but also a distinct group of children whose sole symptoms consist of restlessness, inability to concentrate, impulsiveness, distractibility, and inability to get along with other children, all associated with varying degrees of motor release phenomena and intellectual changes. Occasionally epilepsy appears as well, and actual psychotic symptoms may occur. The young, so-called constitutional schizophrenic mentioned in this paper, and constituting the major portion of the group of schizophrenic psychoses herein described, may belong to this group of "organic" cases.

Jasper[7] and his associates have completed electroencephalographic studies of 71 behavior problem children, 12 girls and 59 boys. Thirty-five had a history suggestive of central nervous system disorders, encephalitis, birth trauma, post-natal trauma, etc. Eleven of the cases were classified as schizoid or schizophrenic. In the entire group of 71 cases, 71 per cent showed abnormalities in brain potential, very marked in 59 per cent, and of the epileptiform variety in 39 per cent. Jasper and his associates conclude that abnormal brain function as revealed by the electroencephalogram is an important component in the etiological picture of the majority of a group of problem children whose disorder had been considered as primarily psychogenic previous to using this new method of diagnosis. While the nature of the fundamental pathology of the brain indicated is not as yet known, the findings have been found important in evaluating prognosis and treatment, according to these authors.

Since the function of the brain cannot be revealed by the electroencephalogram at the present time, one must accept these results and conclusions with certain reservations.

Our so-called "organic" cases suggest a variable type of lesion and are quite confusing from the diagnostic standpoint. Early in life these children may be called defectives, post-encephalitics, or more rarely, pre-schizophrenics. Usually, however, active schizophrenic symptoms begin only when the child is presented with his first socializing influences and is unable to meet the requirements of reality as a result of his many handicaps.

Potter and Klein[8] have classified problem children into an organic reaction group, a situational reaction group, and a psychotic group. In the first they include idiopathic epilepsy, mental deficiency, juvenile paresis, and behavior problems secondary to organic conditions. The psychotic group is concerned chiefly with schizophrenic reactions. These authors reported fourteen such cases and in all of them the onset of the psychosis was before twelve years of age, actually before five years of age in seven. Since only one of the fourteen schizophrenic children improved sufficiently to make an adequate social adjustment, the authors concluded that a constitutional inadequacy factor must be emphasized, whatever it may be.

Sherman[9] recently reported studies of 17 schizophrenic children who were observed for periods of from six months to four years. He points out the great difficulty in recognizing and

defining mental abnormalities in children and the general tendency to pass them off as behavior problems in the broad sense of the word. He justly emphasizes the fact that we are usually dealing with the end-results of a long standing psychotic process when we are confronted with the typical hallucinated and delusional deteriorated schizophrene. He called his children schizophrenics mainly because the pattern of their behavior resembled in many ways the behavior and symptoms of adult schizophrenic patients.

McFarland and Goldstein[10] have summarized the present-day status of biochemical studies in dementia praecox. Quoting them, "The schizophrenic seems to be born tender and exceedingly vulnerable to the 'hurts of the world' and must be protected from the buffets of the environment." They feel that most investigators are of the opinion that the schizophrenic psychosis is a multitude of disorders of various etiologies, and that the schizophrenic reaction is merely a symptom common to this heterogeneous group which includes all causes, physiological, psychological, and others. For that reason they are of the opinion that the biochemistry of schizophrenia may ultimately prove as meaningless and inconclusive as the biochemistry of backache or of abdominal pain, simply because patients have been classified on the basis of reaction-type rather than of etiology. This would apply equally as well to other studies in schizophrenia.

COMMENT

It appears that the so-called schizophrenic syndrome may occur under a large variety of conditions. It is obvious, too, that what we recognize as such a syndrome is relatively rare before puberty and that its incidence rapidly increases from that time to reach a maximum in the middle of the third decade of life. The majority of the cases seen in early childhood appear to belong, generally speaking, to a group characterized by phenomena which seem to be associated with basic organic changes, whatever they may be more specifically. These children appear to be closely related to a type of child, early recognized as a severe behavior problem, and characterized by a symptom complex of hyperactivity, restlessness, asocial tendencies, etc.

Clinical impressions, supplemented by recent electro-encephalographic studies, suggest that there are actual "organic" changes in these cases, and that the total personality is so affected that it is difficult to obtain an adequate social adjustment of the individual with reality and his environment.

It seems that the prenatal and natal influences play an important role in these cases in producing constitutional inadequacies so serious that all subsequent developmental changes are affected.

Our more recently acquired knowledge of the importance of psychosomatic relationships, particularly as they involve the autonomic nervous system and the emotions, furnishes us, however, with the necessity of regarding the first two or three years of life during which the so-called disposition of the individual is molded, as an extremely important period.

Since we are dealing with a fragile, flexible autonomic nervous system in which only postnatally is there actual reflex integration, one may postulate the importance of such psychological factors as early rejection, severe oral thwarting, threatening sexual experiences, punishment and frustration, in contributing to more or less permanent changes in the child's psychobiological life.

It is obvious, therefore, that external influences, both psychological and biological, can affect the future functioning of the individual as a psychobiological unit. Preventive work and mental hygiene activities must therefore concentrate not only on the eugenic aspects of marriage and the great need for careful obstetrical management of every case, but also on that postnatal period just described when a child must develop what Benedek has described as the emotional relationship of "confidence," associated as it is with security, regular satisfactions of individual needs, etc. If these factors are adequately controlled, and if the relatively new psychological knowledge gained through psychoanalysis can be brought into play in the training of the child, there may possibly be fewer schizophrenics, and those cases which do develop later through the appearance of intolerable environmental factors, can then be treated through the removal of the latter, if possible, and then followed up with adequate psychotherapy.

In our group of postpubescent cases psychological factors appeared to be most significant and in these children it seemed that the psychosis occurred when the child was unable to sublimate in a more normal way strong instinctual ten-

dencies which conflicted greatly with a rigid super-ego.

In the prepubescent group it appears that a psychopathic hereditary taint was present, and the psychotic symptoms, when they occurred, seemingly because of psychogenic precipitating factors, were rather bizarre and of an acute nature.

The constitutional group is a motley one. With careful observation on the part of interested individuals, these cases can be recognized earlier than the others and can be followed carefully as a result. It is interesting that many of these children had been under observation at the Institute for years. They were usually able to adjust to some degree, in spite of the recognized peculiarities, until they arrived at late adolescence when it was no longer possible to overlook their need to become social individuals. It then became necessary to consider for them care in a protected environment such as an institution, particularly since these children usually become more withdrawn and develop more autistic symptoms as they grow older. It is interesting to see how many of them continue to attend school for years while presenting definite peculiarities.

I have been obliged to omit the more detailed symptoms of these various schizophrenic children, as well as a consideration of the dynamic factors which appear to indicate an early "split" in them. It is our hope to continue these studies.

SUMMARY

1. A review of the psychotic children seen at the Institute for Juvenile Research during 1938 is presented.

2. The various so-called schizophrenic cases are described and their differences enumerated.

3. The importance of the different etiological factors is postulated for present and future study.

4. There is obviously a need for special facilities for the hospitalization, investigation and treatment of these psychotic children.

BIBLIOGRAPHY

1. Kasanin, J., and Kaufman, M. R.: Am. J. Psych. 9: 307, 1929.
2. Alexander, Franz.: Arch. Neur. and Psych. 26: 815, 1931.
3. Benedek, Therese: Psychoanalytic Quarterly. 7: 200, 1938.
4. Yakovlev, Paul I.: Arch. Neur. and Psych. 41: 119, 1939.
5. Singer, H. Douglas: Jour. A. M. A. 110: 2048, 1938.
6. Peterman, M. G.: Bulletin North Shore Branch Chicago Medical Society, Apr., page 1, 1939.
7. Jasper, H. H., Solomon, Philip, and Bradley, Charles: Amer. J. Psych. 95: 641, 1938.
8. Potter, Howard W., and Klein, Henriette R.: Am. J. Psych. 94: 681, 1937.
9. Sherman, Mandel: Child Development. 10: 35, 1939.
10. McFarland, R. A., and Goldstein, H.: Am. J. Psych. 95: 509, 1938.

DISCUSSION

43 E. Ohio St.

Dr. Maxwell Gitelson, Chicago: I should like to confine my discussion to the constitutional group in which there is evidence of non-integration of the ego, a non-integration of the synthesizing capacity of the patient. This can be subsumed under the statement that it represents a group in which the reality testing ability has failed to reach completeness. If we are to keep this point in mind, then we have a legitimate question as to the ego which has failed to be integrated and as to what happens to it.

We know that the reality testing capacity is not always present. We know that it has to begin in the very earliest phase of our development. The observations that have been made indicate that the reality testing function makes its appearance during the first year of life under the auspices of the maternal care which the infant is at that time of necessity receiving.

To begin with there is no external reality in the sense that we recognize it in life. There is no differentiation between a "me" and a "you." The first differentiation between the "me" and the "you" is, roughly, connected with the weaning from the breast or the weaning from the more intimate relationship with the mother and represents a normal vicissitude in the life of the individual. The point that I am getting at is that the schizophrenias as we usually see them represent a great regression, or breaking down and return to this non-object level of an individual's development. In Dr. Falstein's "constitutional type" the individual has never evolved from his non-object level.

This brings me to the next point in my discussion and this is apropos of the so-called organic factors in the development of schizophrenia. The development of the ego begins at an early period. The integration which the individual acquires in respect to social conduct, those adjustments which he learned through the relationship of security with the mother, his reality testing ability, are educational processes. Where this atmosphere of security and the supplying of the basic needs and basic tendencies is not available, the educational process suffers and the individual acquires his first cognizance of reality as an excessively dangerous thing. This process occurring as early as it does, during the time when organic integration, as we usually understand it, is also going on, is bound to present us with phenomena in which there is an over-lapping of more purely psychic distortions of development and more purely organic distortions of development. I do not believe we are in a clear-cut position to decide that birth injury as such or severe infantile illness as such is the causative factor in the development of "constitutional" schizophrenia, but that they are factors which interfere with the primary educational process, factors which interfere

with the acquisition of integrative and adjustive powers which we call the ego.

With respect to the other types of juvenile schizophrenia in which the ego seems to have been more or less well developed, there are two etiological factors to be considered. The situation which is biologic, which appears at puberty with an exacerbation of the endocrine functions, and secondly, the situations which are connected with actual dangerously seductive relationships with individuals in the world at large. For instance, I recall the case of a young schizophrenic who from birth had been exposed to such a seductive relationship with his psychotic mother who was able to resume a relatively normal development by the procedure of separation from the mother.

Schizophrenia may be the consequence of a weakening of a relatively intact or well developed ego by exogenous factors such as intoxications, injuries or other similar organic disturbances. The point that Jasper has made about the evidence of organic disturbance in the brain on the basis of his encephalographic findings explaining behavior defects in children needs to be questioned because with the possible exception of the encephalogram of epilepsy, the finding may be reversible. I would like to state that data are being gathered by Dr. Leon Saul at The Institute for Psycho-Analysis showing that during the analysis of neurotic patients encephalographic waves have changed.

A propos of the previous discussion of general trauma and head injury, we have within the last year at the Institute for Juvenile Research had the experience of having a case sent in by a family for examination, found the child feeble-minded, so stated to the parents, and advised commitment. This was followed by a letter from the family lawyer asking us to testify to our findings in a suit that the parents had entered against an automobile driver for injury to the child. Fortunately in this case we had examined the child some years before the injury and knew the child's feeblemindedness to have antedated the accident, but in a number of less clear-cut cases similar problems have arisen. So I should like to emphasize the necessity of a careful study of the character of the preceding personality in respect to feeblemindedness and previous psychotic and neurotic disturbances before connecting the final picture with trauma.

CARCINOMA OF RECTOSIGMOID IN PATIENT TWENTY-SIX YEARS OF AGE; SIX-YEAR CURE FOLLOWING ABDOMINAL RESECTION

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A male, 26 years of age, a shipping clerk by trade, entered St. Luke's Hospital March 3, 1933, complaining of constipation of eight months' duration. For six months he occasionally had noted bright red blood in the stool, with paroxysms of diarrhea for four months, followed by constipation, and the loss of ten pounds, which was attributed to a lack of appetite by the patient. The family and past histories were essentially negative.

Physical examination revealed a well-developed young male weighing 150 pounds. The erythrocytes numbered 4,120,000 per cu. mm., the leucocytes 8,350 per cu. mm., and the hemoglobin determination revealed 15.5 gms. per 100 cc. The differential blood count was normal. The urine examination was negative. The blood pressure was 112/68. A proctoscopic examination revealed an annular obstructing lesion at the rectosigmoid junction. A portion of the tumor removed for microscopic study was reported to be adenocarcinoma. The patient was put on a low residue diet for 72 hours and the lower bowel thoroughly emptied by repeated enemata.

Under ethylene anesthesia, a low mid-line incision was made. Just distal to the peritoneal reflection was a napkin ring obstruction; the opening in the tumor admitted only the tip of the index finger. No palpable regional lymph nodes were found. There was no evidence of metastasis in the liver.

Through a left muscle-splitting McArthur incision, the colon was exteriorized for a colostomy. The colon was opened with an electrocautery at the end of 48 hours. The course was uneventful and the lower loop was irrigated regularly with normal saline solution.

Ten days following the abdominal exploration, the patient again was operated upon. Under ethylene anesthesia the midline incision was reopened. The tumor mass was resected and an end to end anastomosis made. A Penrose drain was inserted just below the peritoneal reflection. The postoperative course was smooth and the patient was discharged fifteen days following the second operation.

The pathological examination revealed that 10 cm. of bowel were resected with the tumor lying midway between the two ends. The lumen of the bowel at the point of construction measured 1 cm. Two lymph nodes contained metastatic carcinoma. The patient was given Roentgen therapy the succeeding three months and has been observed at frequent intervals during the past five years. At the time of writing, he was at his peak weight of 180 pounds; he stated he

"felt better than at any other time" in his life. His bowel habits were regular. Proctoscopic examination revealed a slight constriction at the site of anastomosis.

DISCUSSION

This case is of interest because of the age of the patient, he being 26 years old. Carcinoma occurring under the age of 35 years presents an extremely poor outlook. Five-year cures are not common and many patients are unoperable due to early metastasis by the time the surgeons is consulted. In the past ten years, we have had four cases of carcinoma of the rectum and sigmoid in patients under the age of 30 years; one, 22 years of age, male; two 26 years of age, both males; and one 28 years of age, female. The one at the age of 22 years, had generalized abdominal metastasis under exploration and lived 18 months following colostomy. This case was our second. The third patient, also at the age of 26 years, had generalized abdominal metastasis and lived 14 months following colostomy. The fourth, 28 years old, was resected. She was alive after four and one-half years and apparently free from any recurrence. It was interesting that she had formed an iliosigmoidostomy. In our clinic, fifty per cent. of young patients with malignancy of the resto-sigmoid under 30 years of age have had successful end results. Such favorable results, however, should not be anticipated in any sizable series of cases.

The type of resection made is also of interest. According to Miles, carcinoma of the rectum spreads by lymphatic extension downward as well as upward and, therefore, involves the sphincter structures, the perineal skin and the ischorectal spaces. We have never observed this downward spread nor the involvement of these structures except by direct extension. In other words, a growth to involve these structures must be, primarily, very low. For several years we have been impressed with the infrequency of these findings and, except for the very low growths that involve the perineal structure by direct extension, have not observed such downward extension. Probably many perineums are being sacrificed without any particular advantage.

According to Westheus, a German surgeon, and David and Gildchrist, metastatic development from carcinoma of the recto-sigmoid region occurs cephalward and very rarely distally. When it does occur distally the nodules are found in near proximity to the parent tumor. Thus it would seem that it would be sufficient to amputate the bowel approximately five centimeters below the parent tumor. If this can be done and the blood supply of the bowel will permit restoring continuity of the bowel, it would seem advisable to do so in selected cases.

This particular growth, as pointed out (E.L.S.) was mainly below the peritoneal reflection; therefore, the lower segment of the bowel, after extirpation of the tumor, was devoid of serosa. This causes a rather difficult anastomosis as sutures do not hold well and care must be exercised to avoid tension. Infection is not a serious problem because sufficient time is allowed to elapse following primary colostomy. The isolated bowel becomes surgically clean or void of pathogenic bacteria; approximately three weeks are sufficient for this to occur. Irrigations and mild antiseptics, such as boric acid or from one to five thousand potassium permanganate, may be administered. The pelvic peritoneum is closed about the bowel and above the site of the completed anastomosis. A small cigarette drain is placed to the site of the anastomosis and brought out the anterior abdomen.

Microscopic study of this specimen revealed cancer cells in the perirectal fat, as well as lymph-node involvement. The question as to whether or not all cells had been removed became very important. The feeling was that they probably had not been. Roentgenotherapy, therefore, was decided upon. Dr. E. L. Jenkinson, radiologist at St. Luke's Hospital, always wary of treating patients in whom bowel and anastomosis recently had been done, felt that if sufficient therapy were had to destroy cancer cells, the anastomosis may break down. This thesis was well demonstrated in this case. About three months were allowed to elapse before therapy was begun. During the process of therapy, however, the mucous membrane, involving about one-half the circumference of the bowel, broke down. This required about six months to heal again. No complications, fortunately, arose. It is not known how much credit is due Roentgen therapy in this patient nor can we compare, for our resected malignancies are not treated routinely.

THE EFFECTS OF SMOKING*

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The present paper is a report of experiments undertaken in the hopes of explaining the nature of the hold which tobacco has upon its habitual users—why they feel so badly when they smoke and why they feel so much worse when they stop smoking.

There is ample evidence apart from that supplied by experimental methods that tobacco contains drugs which are habit forming. Most persons who smoke, if they are quite frank, realize that it is bad for them, and wish frequently that they were rid of the habit. Most, too, make frequent ineffectual attempts to stop, and cut down or stop entirely, but then drift gradually back, realizing that the thing has them and that they cannot afford the months of discomfort and depression incident to the cure. Admitting their defeat, they content themselves with advising others not to start.

Further evidence is the strange industry with which the smoker goes about finding tobacco or a match when the time comes for him to smoke. One would walk more than a mile for either of them.

Before proceeding, I should remark that the statements made in this paper hold only for those who inhale tobacco smoke. The habit is rarely formed by those who do not draw the smoke into their lungs and the physiological effects here noted are not produced. The lungs are one of the largest and most important absorbing surfaces of the body and it has been proved experimentally that drugs injected into them are absorbed more rapidly than when introduced into the body by any route save that of the blood stream.

Further evidence in support of the contention that tobacco contains habit forming drugs is the fact that a tolerance can be acquired for it, that eventually the smoker can take doses which if consumed when he was first starting to smoke would have been certainly fatal. In my own case, my first essays at smoking when a boy were followed by severe prostration, terrible nausea, and worse vomiting. Determined to be a man, I persisted and gradually acquired a tolerance. In spite of this, for many years, if I smoked a cigar or inhaled too many cigarettes, I became sick. I had to watch the dose. That is all past now and I can smoke any number of cigarettes or cigars, and can chew tobacco and swallow the juice without suffering worse consequences than a tiredness and laziness the following day.

I became curious, as who would not, realizing that smoking kept me from normal efficiency, why I was unable to stop, why I felt so badly when I smoked, and why I felt so much worse when I did not; why I had to smoke as soon as I rose in the morning and before I felt awake and ready for the day, why I had to go out to smoke between acts at the theatre, why in fact I was such a slave to so pleasant and miserable a habit.

Ordinarily I smoke ten to twenty cigarettes a day. If I smoke the latter number for a week or more I become tired, irritable, and nervous, and am troubled with insomnia. These symptoms become so bothersome that I am led to cut down to ten or less a day but then, feeling better, I gradually relax the effort that this entails and am soon back at my maximum and am again soon warned to cut down. I do not smoke before breakfast, but I go down to eat before shaving so that I can get in my smoke without delay. The first morning cigarette occasionally makes me slightly dizzy and until I have finished it I do not feel awake or ready for the day. During the first hour after breakfast I usually smoke three or four cigarettes. After that I smoke whenever I am not doing something that makes it impossible. I smoke between courses at meals and rush my dessert so that I can have my cigarette. The desire seems greatest after a meal. Smoking just before a meal cuts down my appetite noticeably. I do not, as some do, wake up in the middle of the night to smoke. If I am nervous about anything or have any ordeal to go through, I smoke one cigarette after another.

The pleasure of smoking is, like drinking water when thirsty, a relief from the discomfort

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This paper was written in the year 1930 and presented before the Evanston Branch of the Chicago Medical Society in May, 1931. I have heretofore refrained from publishing it, feeling that observations made on a single case were not sufficiently conclusive. Although the whole subject of the effects of tobacco has been extensively investigated since this paper was prepared, on re-reading it lately it seemed to me that it was valuable in its description of the symptoms of tobacco deprivation and that the physiological findings were sufficiently suggestive to warrant further investigation.

that rapidly develops if I do not smoke. The nature of this discomfort has been rather difficult to analyze. Unless one regards it closely it is recognized merely as a desire to smoke. About an hour after I have had a cigarette I develop a peculiar sensation in my mouth. I cannot tell whether it is a dryness or a wetness or what it is, but my tongue and lips and cheeks come into my consciousness and seem swollen and burn slightly. I feel like sucking my cheeks, biting my lips, and scraping my tongue over my teeth. I experience a metallic taste. Sucking pebbles or hard candy or chewing gum afford considerable relief. This increases for an hour or two and then remains at its maximum for at least two months, which is as long as I have ever refrained from smoking. As it becomes more severe there develops a sense of tightness in the face. This and the other mouth sensations are, as the experiments to be reported indicate, probably incident to an overactivity of the salivery glands.

During the second hour and soon after the appearance of the mouth symptoms, I begin to be conscious of my legs and arms—in fact of my whole body. It is difficult to describe these sensations accurately, for there is no other feeling like it. It is not so much a pain as an awareness of every muscle and nerve in the body. This is the true urge to another cigarette and disappears almost with the first puff. It is very pleasant to be relieved of it. Coincident with the appearance of the other symptoms my respirations become slow and irregular. Typical vagal sighs are frequent.

As the desire to smoke increases I become mentally dull. I cannot work or concentrate. If I keep awake I cannot sit still but have to walk about or do something. All that I can think of is smoking. I can go to sleep easily, much more easily than when I am smoking, and sleep is desirable as a relief from the discomfort. Active physical exercise, such as brisk walking, relieves most of the symptoms.

It has long seemed certain to me that these severe subjective symptoms must be manifestations of definite physiological alterations. To determine what these were, I studied the effect of smoking upon the pulse, the respirations, the blood pressure, the basal metabolism, and the rate of salivary excretion.

On rising in the morning after eight hours of deprivation, I found that my pulse and respirations were slow, my blood pressure and basal metabolism low and my salivary secretion markedly increased. At this time of day and continuing until my first cigarette my pulse varies between 55 and 60 beats per minute and my respirations between 12 and 14. My blood pressure runs from 90 to 100 systolic and 60 to 65 diastolic; my basal metabolism is —10 to —5 and my salivary excretion 5 cc. in three minutes. I feel apathetic and mentally dull and irritable.

By the time I have finished inhaling a cigarette—almost immediately—all of these factors return to normal. My pulse rises to 84, my respirations to 20, my blood pressure to 120/75, my basal metabolism to Plus 4 or Plus 5, and the flow of saliva is stopped almost completely. At the first few puffs my head swims a little, but by the time I have finished the cigarette this has passed, my mental depression has disappeared and I feel awake and ready for the day. I almost immediately have the urge to defecate and do this promptly, rapidly, and satisfactorily.

If I smoke only the one cigarette, all of the factors observed remain at their new level for approximately an hour, but then during the second hour begin to fall off, the pulse and respiration first, and the blood pressure later. The onset of these changes coincides with the appearance of the desire to smoke and the desire increases commensurately with the changes. The changes in the respiratory rate and rhythm are very noticeable. I breathe slowly and deeply and have occasional deep sighing respirations. By the end of two hours I am decidedly uncomfortable and the readings have all approached the pre-smoking level. If, at this time I smoke another cigarette, they all return rapidly to normal as they did earlier.

If I smoke every two hours during the day this same cycle is repeated during each period and the curve for the day shows a series of rapid rises and gradual falls.

If I smoke every hour during the day there are slight falls towards the end of the first two hours but after that the pulse, blood pressure and respirations remain at a constant normal level. Smoking more frequently does not further elevate them yet I have a feeling that towards

the end of the day I smoke more often and with less enjoyment than in the morning. I believe, although I have not proved it, that this is because the nervous system has become tired from repeated stimulations. It is probable that this also explains the increase in the general physical weariness which approaches towards evening and which is greater on days during which I have smoked excessively.

These findings suggest that inhaling tobacco smoke stimulates the sympathetic nervous system, as opposed to the parasympathetic, much as does adrenalin, and that eventually these nerves become dependent upon the drug to maintain them in normal tone and to keep them in balance with the parasympathetics. When the habitual smoker refrains from smoking his sympathetic nervous system, dependent on the drug for normal function, becomes depressed and the parasympathetics assume control. He becomes definitely vagotonic. He must smoke to remain normal.

As has been stated, the phenomena studied, the pulse, respirations, blood pressure, basal metabolism, and salivary excretions, all indicate that cessation of smoking induces a predominance of the parasympathetic nerves over the sympathetic. If this is so, then all of the other vegetative functions are altered by smoking or by refraining from smoking. Tobacco deprivation must produce an increased secretion of gastric juice, an increased peristalsis, a tightening of the gastrointestinal and urinary bladder spincters, an increased secretion of insulin, a lowering of blood sugar and so on and so forth. On the other hand, smoking must cause a decrease in the secretion of gastric juice, a cessation of peristalsis, in fact the reverse of all the states produced by not smoking.

These changes explain why smoking immediately before a meal destroys the appetite, by cutting down the psychic flow of saliva and gastric juice and this loss of appetite helps to explain the loss of weight incident to smoking and the rapid gain on stopping. Stimulation of the sympathetics accounts for the prompt appearance of the desire to defecate after the first morning cigarette, and preponderance of the parasympathetics, the tendency to rectal constipation and incomplete defecation associated with refraining from smoking.

The effects on the entire cardiovascular system can easily be predicated.

That smoking causes a stimulation of the sympathetics and not smoking a domination of the parasympathetics is not entirely a justifiable conclusion. The phenomena observed can as well be interpreted in the opposite sense, that tobacco causes a depression of the parasympathetics, which, upon the habitual use of the drug, adapt themselves to withstand it and maintain their function under its effects, and upon cessation of its use become overactive.

Granting that smoking is a physiological habit, it is important to know how strong a habit it is. Bert Leston Taylor used to speak of it and defend it as one of the minor vices; and that it is not a bad one is argued by the relative impunity with which it is indulged in by so large a proportion of the population.

As vices go, it is probably comparable to the common cold among illnesses. But the common cold, while not threatening life, is, because of its prevalence, a very important disease, accounting in the aggregate for a tremendous loss of working time and a still greater loss of working efficiency. Smoking similarly, while rarely causing serious consequences, certainly is a bad thing for the individual and for the race.

It is not an especially strong habit. While it is uncommon to find anyone who inhales who stops permanently, many do stop for long periods. Those who stop permanently do so usually because they have been frightened by symptoms attributed to smoking. Deprivation causes discomfort but no serious manifestations.

In my own case, I can bring myself to undergo the discomfort incident to stopping only by imposing upon myself some forfeit for breaking my resolution. I can do this best by betting with some one else that I will not smoke for a given period. In this case pride and the unwillingness to lose the bet are sufficient to hold me to my purpose. I recently stopped for two months. I did not, during this time, keep records of the phenomena observed in the shorter experiments and consequently can only report on my subjective symptoms and the more obvious physiological phenomena.

After the first day, and as soon as I had convinced myself that I would not give in to my desire, the discomfort was less bothersome. It was not absent, but I simply went about my

that rapidly develops if I do not smoke. The nature of this discomfort has been rather difficult to analyze. Unless one regards it closely it is recognized merely as a desire to smoke. About an hour after I have had a cigarette I develop a peculiar sensation in my mouth. I cannot tell whether it is a dryness or a wetness or what it is, but my tongue and lips and cheeks come into my consciousness and seem swollen and burn slightly. I feel like sucking my cheeks, biting my lips, and scraping my tongue over my teeth. I experience a metallic taste. Sucking pebbles or hard candy or chewing gum afford considerable relief. This increases for an hour or two and then remains at its maximum for at least two months, which is as long as I have ever refrained from smoking. As it becomes more severe there develops a sense of tightness in the face. This and the other mouth sensations are, as the experiments to be reported indicate, probably incident to an overactivity of the salivery glands.

During the second hour and soon after the appearance of the mouth symptoms, I begin to be conscious of my legs and arms—in fact of my whole body. It is difficult to describe these sensations accurately, for there is no other feeling like it. It is not so much a pain as an awareness of every muscle and nerve in the body. This is the true urge to another cigarette and disappears almost with the first puff. It is very pleasant to be relieved of it. Coincident with the appearance of the other symptoms my respirations become slow and irregular. Typical vagal sighs are frequent.

As the desire to smoke increases I become mentally dull. I cannot work or concentrate. If I keep awake I cannot sit still but have to walk about or do something. All that I can think of is smoking. I can go to sleep easily, much more easily than when I am smoking, and sleep is desirable as a relief from the discomfort. Active physical exercise, such as brisk walking, relieves most of the symptoms.

It has long seemed certain to me that these severe subjective symptoms must be manifestations of definite physiological alterations. To determine what these were, I studied the effect of smoking upon the pulse, the respirations, the blood pressure, the basal metabolism, and the rate of salivary excretion.

On rising in the morning after eight hours of deprivation, I found that my pulse and respirations were slow, my blood pressure and basal metabolism low and my salivary secretion markedly increased. At this time of day and continuing until my first cigarette my pulse varies between 55 and 60 beats per minute and my respirations between 12 and 14. My blood pressure runs from 90 to 100 systolic and 60 to 65 diastolic; my basal metabolism is —10 to —5 and my salivary excretion 5 cc. in three minutes. I feel apathetic and mentally dull and irritable.

By the time I have finished inhaling a cigarette—almost immediately—all of these factors return to normal. My pulse rises to 84, my respirations to 20, my blood pressure to 120/75, my basal metabolism to Plus 4 or Plus 5, and the flow of saliva is stopped almost completely. At the first few puffs my head swims a little, but by the time I have finished the cigarette this has passed, my mental depression has disappeared and I feel awake and ready for the day. I almost immediately have the urge to defecate and do this promptly, rapidly, and satisfactorily.

If I smoke only the one cigarette, all of the factors observed remain at their new level for approximately an hour, but then during the second hour begin to fall off, the pulse and respiration first, and the blood pressure later. The onset of these changes coincides with the appearance of the desire to smoke and the desire increases commensurately with the changes. The changes in the respiratory rate and rhythm are very noticeable. I breathe slowly and deeply and have occasional deep sighing respirations. By the end of two hours I am decidedly uncomfortable and the readings have all approached the pre-smoking level. If, at this time I smoke another cigarette, they all return rapidly to normal as they did earlier.

If I smoke every two hours during the day this same cycle is repeated during each period and the curve for the day shows a series of rapid rises and gradual falls.

If I smoke every hour during the day there are slight falls towards the end of the first two hours but after that the pulse, blood pressure and respirations remain at a constant normal level. Smoking more frequently does not further elevate them yet I have a feeling that towards

the end of the day I smoke more often and with less enjoyment than in the morning. I believe, although I have not proved it, that this is because the nervous system has become tired from repeated stimulations. It is probable that this also explains the increase in the general physical weariness which approaches towards evening and which is greater on days during which I have smoked excessively.

These findings suggest that inhaling tobacco smoke stimulates the sympathetic nervous system, as opposed to the parasympathetic, much as does adrenalin, and that eventually these nerves become dependent upon the drug to maintain them in normal tone and to keep them in balance with the parasympathetics. When the habitual smoker refrains from smoking his sympathetic nervous system, dependent on the drug for normal function, becomes depressed and the parasympathetics assume control. He becomes definitely vagotonic. He must smoke to remain normal.

As has been stated, the phenomena studied, the pulse, respirations, blood pressure, basal metabolism, and salivary excretions, all indicate that cessation of smoking induces a predominance of the parasympathetic nerves over the sympathetic. If this is so, then all of the other vegetative functions are altered by smoking or by refraining from smoking. Tobacco deprivation must produce an increased secretion of gastric juice, an increased peristalsis, a tightening of the gastrointestinal and urinary bladder spincters, an increased secretion of insulin, a lowering of blood sugar and so on and so forth. On the other hand, smoking must cause a decrease in the secretion of gastric juice, a cessation of peristalsis, in fact the reverse of all the states produced by not smoking.

These changes explain why smoking immediately before a meal destroys the appetite, by cutting down the psychic flow of saliva and gastric juice and this loss of appetite helps to explain the loss of weight incident to smoking and the rapid gain on stopping. Stimulation of the sympathetics accounts for the prompt appearance of the desire to defecate after the first morning cigarette, and preponderance of the parasympathetics, the tendency to rectal constipation and incomplete defecation associated with refraining from smoking.

The effects on the entire cardiovascular system can easily be predicated.

That smoking causes a stimulation of the sympathetics and not smoking a domination of the parasympathetics is not entirely a justifiable conclusion. The phenomena observed can as well be interpreted in the opposite sense, that tobacco causes a depression of the parasympathetics, which, upon the habitual use of the drug, adapt themselves to withstand it and maintain their function under its effects, and upon cessation of its use become overactive.

Granting that smoking is a physiological habit, it is important to know how strong a habit it is. Bert Leston Taylor used to speak of it and defend it as one of the minor vices; and that it is not a bad one is argued by the relative impunity with which it is indulged in by so large a proportion of the population.

As vices go, it is probably comparable to the common cold among illnesses. But the common cold, while not threatening life, is, because of its prevalence, a very important disease, accounting in the aggregate for a tremendous loss of working time and a still greater loss of working efficiency. Smoking similarly, while rarely causing serious consequences, certainly is a bad thing for the individual and for the race.

It is not an especially strong habit. While it is uncommon to find anyone who inhales who stops permanently, many do stop for long periods. Those who stop permanently do so usually because they have been frightened by symptoms attributed to smoking. Deprivation causes discomfort but no serious manifestations.

In my own case, I can bring myself to undergo the discomfort incident to stopping only by imposing upon myself some forfeit for breaking my resolution. I can do this best by betting with some one else that I will not smoke for a given period. In this case pride and the unwillingness to lose the bet are sufficient to hold me to my purpose. I recently stopped for two months. I did not, during this time, keep records of the phenomena observed in the shorter experiments and consequently can only report on my subjective symptoms and the more obvious physiological phenomena.

After the first day, and as soon as I had convinced myself that I would not give in to my desire, the discomfort was less bothersome. It was not absent, but I simply went about my

business and frequently forgot it. I was more comfortable when walking or exercising strenuously or doing anything that tends to raise the blood pressure. The discomfort and desire was not constant. On some days I would notice them very little and again they would come back as strongly as ever and for long periods the thought of smoking was constantly in my mind. Waves of desire to smoke were associated usually with lack of sleep or worry. The last week of the two months was the most difficult for the thought that I would soon be able to smoke was constantly with me.

Throughout the two months I kept a piece of hard candy in my mouth most of the time. The oral discomfort persisted throughout the period. My appetite was better than it had been since I can remember. I looked forward to and enjoyed my meals, a thing I rarely do when smoking. During the two months, I gained 15 pounds and was still gaining when the time came to smoke again. I slept well and was more rested when I wakened in the morning. When I am smoking I am occasionally troubled with insomnia. This did not bother me and in the morning, instead of finding it almost impossible to get up, I wakened sharply and felt refreshed and liked rising immediately.

Throughout the two months my bowels were sluggish and irregular, and defecation incomplete and unsatisfactory. I rarely had the urge to move my bowels and when I did it was not at the habitual time.

By the end of a week I noticed that cigarettes and tobacco in general had an odor that I had not associated with them since boyhood. I had always believed that cigarettes were different then, but realize now that saturation with tobacco smoke has made me insensitive to their sweetness and aroma.

One of the striking phenomena incident to not smoking was my reaction to coffee. Coffee usually makes me nervous and a single cup after four in the afternoon will keep me awake far into the night. There are times when I am smoking much that even a single cup in the morning makes me nervous and excited until noon. During the two months that I was not smoking, I could drink as much coffee as I wished without becoming nervous, and two cups at supper and one late in the evening failed to keep me from sleeping.

Finally the great day arrived and I hurried through my breakfast and sighed with relief as I lit a cigarette. I was to be normal again. I was unpleasantly surprised. Not only did the smoke taste like that from an old rag but on attempting to inhale I was choked up as I had been when I was learning. It was evident that I would have to learn to smoke again. After taking a few puffs I threw the cigarette away and went to the store and bought a package of de-nicotinized cigarettes. These, although they tasted bad, I could inhale and doing this lustily I went about my work. The second surprise followed quickly. I found myself nauseated—truly sick, as I had not been for years—and I had to spend the morning in the rest room and in close proximity to the toilet.

That was three months ago. By gradually increasing the dose I have again become a smoker and can inhale regular cigarettes like a regular fellow.

It will seem strange to many that having stopped for two months one should return to a habit which he realizes is unnatural and deleterious. I returned simply because I could no longer afford to be as inefficient and as disturbed psychologically as I was when refraining. It is my belief that the most serious and important effects of not smoking are manifested in changes in personality. Smoking, as we have seen, stimulates the sympathetic nervous system and, I am sure, has certain sedative effects upon the cerebrum. The sympathetic nervous system controls the function of the glands of internal secretion and these in large part determine one's personality. When one smokes he is content to sit and do nothing else. He is stimulated physically and soothed mentally. Pressing problems and the worry and perplexity of existence can be put off momentarily while one withdraws behind his smoke screen. When he comes out he is perhaps a little better able to deal with them. He is keyed up and the mind is in a state less easily daunted. However this works, it is certain that smoking weaves itself into one's whole scheme of life. It is a pacifier, a stimulant, a temporary retreat. One's whole physiology and psychology become dependent upon it. To stop is not only to alter one's basic physiology but one's whole personality, his whole method of reacting to his surroundings. One can no longer relax momentarily and escape, nor

stimulate himself to meet problems, or dull himself to withstand their onslaught. He is a different person, strange to himself, and life is different. To expect one to change 20-year-old habits of physiology is not too much, but to expect him to change psychological habits of this long standing, to alter his whole scheme of reacting to his surroundings is to expect more than is just or reasonable. Most prefer to go on with themselves as they have been rather than start anew with a personality that is strange and which may be, after all, less efficient and less satisfactory than that moulded for them by the drug. So they keep on smoking, but they advise others not to start.

Tobacco contains drugs which have a powerful effect upon the sympathetic nervous system, drugs which are definitely habit forming. That smoking is a vice is certain but that, as B.L.T. has said, it is a minor vice, is equally sure, for most of us smoke immoderately and yet work hard and effectively and live long. It is even conceivable that smoking accounts in part for our ability to meet the increasing strain of business life without breaking. It is probable that for certain psychological types it is more beneficial than harmful. I feel that the psychiatrists would do well to investigate this aspect of the drug and perhaps prescribe it for their vagotonics.

What role it has in producing disease is uncertain. To find that the blood pressure of smokers is not higher than that of non-smokers means this and nothing else. It does not mean that the repeated rises of blood pressure, the repeated lashes of the sympathetic nervous system do not affect the coronary vessels and in the long run lead to sclerosis and occlusion. It is quite conceivable that they do, and no one is justified in saying that the increase of sudden cardiac deaths in men under 60 is not bound up with the recent increase in cigarette smoking. We know that cigarette smoking plays a role in the etiology of Buerger's disease and there is reason to believe that it should do some permanent damage to the whole peripheral vascular system. It must be definitely injurious to the sympatheticotonic personality.

All of these questions are important, are undecided, and are extremely difficult to investigate. That they should be investigated carefully by the medical profession or the public health service is obvious. It is an anachronism that as a people we should allow the cigarette manufacturers to entice young men and young women to develop the habit of using so strong a drug without knowing more about the effects of its use.

SOME EFFECTS OF THE INJECTION OF PITRESSIN IN DEMENTIA PRAECOX

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Evidence of dysfunction of the vegetative nervous system in dementia praecox has been reported by many workers. However, it is not known whether there is a deficient reactivity of the peripheral portion of the vegetative nervous system, in the center, or both. A study of the effects of a drug which acts on the effector organs innervated by the vegetative nervous system should help in partly clarifying this problem. For this purpose we used pitressin. Pitressin is an active principle of the posterior pituitary gland which stimulates intestinal peristalsis, reduces kidney excretion and is said to cause a rise in blood pressure.

The effects of the injection of pitressin was studied in 15 acute schizophrenic patients, five patients with manic-depressive psychosis, four patients with epilepsy and five normal subjects. Ten units of pitressin were given intramuscularly and the blood pressure and pulse were taken every two minutes for thirty minutes. Due to the vasoconstrictor effects of pitressin the skin of the face was markedly blanched but no untoward effects occurred. In many of the subjects these tests were repeated for several days. Blood sugar levels were also determined before and every fifteen minutes for ninety minutes after the injections of pitressin. There was a transient rise in blood pressure five minutes after the injection for an average of 10 mm. of mercury, systolic and diastolic in ten schizophrenics. In three there was no change and in two it was lowered. This slight pressor re-

We are indebted to Parke-Davis & Co. for the pitressin used in these experiments.

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sponse, and at times a lowering of blood pressure, was also observed in the normal subjects and in the patients with epilepsy. In manic-depressive psychosis, however, there was a rise of about 20 mm. of mercury systolic and diastolic and a lowering of the blood pressure was not observed. The effect on the pulse rate was also similar in schizophrenics, normal subjects and epileptics. In each group there were equal instances of a rise, diminution or no change in the pulse rate. In the patients with manic-depressive psychosis, the pulse rate was always lowered. Blood sugar curves were obtained only in dementia praecox and in manic-depressive psychosis. In each group there was more often a hyperglycemic response and in a few instances no change.

The most striking response to pitressin observed was the severe intestinal cramps, intense desire for defecation and successful expulsion of the contents of the colon in each of the normal subjects a few minutes after the injection of pitressin. The patients with schizophrenia neither complained of intestinal cramps nor was distress indicated in their facial expressions. The contents of the colon were not expelled for many hours after the injection. In the patients with epilepsy and manic-depressive psychosis we failed to make observation for this effect of pitressin. In view of this important finding on the failure of pitressin to cause peristalsis in dementia praecox, the problem will be studied on a larger scale with the aid of x-rays and kymographic tracings. Prostigmine will also be used.

The failure of pitressin in causing motility of the colon is of interest in view of the report by Henry of decreased motility and retention of barium in the colon in acute schizophrenia. He interprets this as due to predominance of the sympathetic division with a cessation of activity of the parasympathetic and, therefore, an inhibition of digestive processes.

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REFERENCES

Henry, Geo. W.: Gastrointestinal Motor Functions In Schizophrenia, Assn. for Research in Nervous and Mental Diseases. 5: 280, 1928. (Paul B. Hoeber, Inc.)

ENDEMIC TYPHUS FEVER

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Endemic typhus fever has been recognized in parts of the United States for many years. Since first observed by Brill in New York in 1897, it has been known to be present along the southern and eastern seacoasts from the Mexican border to New England. In central and western United States it has been and continues to be a relatively rare disease. Its occasional occurrence however, necessitates its consideration in certain cases of otherwise unexplained fever.

Endemic or New World typhus is not to be confused with epidemic or Old World typhus fever. The latter is an ancient disease, highly infectious and associated with a high mortality rate. It thrives in filthy, overcrowded surroundings and is transmitted by the human body louse. On the other hand, endemic typhus fever is usually mild, associated with a low mortality rate and is thought to be transmitted to man from a rodent reservoir by the rat flea.[1] The diagnosis of epidemic typhus fever during an outbreak offers no difficulty. Endemic typhus occurring in its usual localities is also generally recognized, but when it appears in regions where it rarely occurs, as it may when individuals are travelling across the country from prevalent to non-prevalent areas, it leads to complications in the usual diagnostic procedures.

CASE

Miss L. G., a twenty-one year old university student, was admitted to the Evanston Hospital on January 6, 1938, and discharged on January 22, 1938. She had been perfectly well until two days before entering the hospital when her illness began. Her complaints on admission were fever, generalized aching particularly of the extremities and back with headache and malaise. The past history, family history and social history were essentially negative except for a trip to Florida during the Christmas holidays.

Physical examination revealed a well-nourished, well-developed girl with a flushed face, slightly reddened throat and a short systolic murmur at the apex. All findings were otherwise normal except for a temperature of 101.4° F., and a pulse of 94. The urinalysis

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was negative, the leucocyte count 8,500 and the red cell count normal. A tentative diagnosis of an influenzal type infection was made giving consideration to the possibility of acute rheumatic fever, undulant fever and rheumatoid arthritis.

For the first two hospital days the patient was treated symptomatically after which all discomfort disappeared. After having dropped to normal on the second day, the temperature began a step-like rise, reaching 103.8° F., on the sixth hospital day, the pulse remaining relatively slow, 90 to 100. Physical findings continued to remain essentially negative.

During this period blood cultures, agglutination tests for typhoid, paratyphoid, undulant fever and tularemia were negative. No malarial parasites could be found on several examinations. The red-cell count, hemoglobin, leucocyte count and differential remained within normal limits. Roentgenograms of the chest were negative as were the Kahn, formol gel and the Forsman heterophile antibody tests. The sedimentation rate was elevated to 32 mm. in 45 minutes.

On the tenth day of the illness, with the fever still 103° F., a rash was first noted on the upper abdomen, macular in type and not associated with itching. The lesions were small, 1 to 3 mm., discrete and reddish in color and were scattered over the abdomen, chest, neck, back and the medial surfaces of the upper extremities and thighs.

The appearance of a rash late in the course of the fever suggested the consideration of illnesses in which a rash appears several days after the onset of the fever. Because of the patient's recent visit to Florida, diseases prevalent along the southeastern seacoast and rare in this part of the country such as dengue, typhus and Rocky Mountain spotted fever were included in the differential diagnosis. A Weil-Fleix agglutination titre with proteus X 19 was found to be positive in a dilution of one to 1,280, the highest titre set up by the bacteriologist at that time. The test was repeated at the Northwestern University Medical School where the agglutination titre proved positive with proteus X 19 in titre of one to 5,120, with proteus OXK in one to 2,560 and with proteus OX 19 one to 2,560. When repeated one month later, these titres were found to be the same. Dr. A. A. Day[2] of the bacteriology department was of the opinion that agglutination in such high titres was unusual and signified typhus rather than spotted fever.

Within three or four days the rash had disappeared; the temperature gradually returned to normal and on the nineteenth day of her illness, the patient was discharged with a diagnosis of endemic typhus fever.

DISCUSSION

Within the past two years no other case of typhus fever has been reported to the Illinois State Health Department.[3] It is of interest to note that prior to 1936 several cases of typhus fever but practically no cases of Rocky Mountain spotted fever had been reported annually. Conversely, since 1936, several cases of the latter and none of the former have been reported. Due to the similarity of the two diseases, it appears that what had formerly been called typhus fever may in reality have been spotted fever, and it suggests that the incidence of typhus in Illinois is less than that indicated by statistics. These two Ricketsia diseases are much alike in onset and course but certain essential clinical differences have been carefully described by Rumreich[4] and others. In general, the fever, symptoms and mortality of spotted fever are more severe. In both diseases the rash appears on or about the fifth day, tending to appear earlier in spotted fever. In typhus the rash is most prominent on the lower chest and upper surfaces of the arms and thighs; the palms, soles and face are rarely involved. In spotted fever the rash is first noticed and is most prominent on the wrists and ankles, spreading in centripetal fashion and may become generalized. Unlike typhus, the palms and soles are usually and the face frequently involved.

The Weil-Felix titre with proteus X 19 is positive in both diseases but most authorities agree that the higher titres are found only in typhus fever. According to Topley and Wilson[5] the Weil-Felix titre in Rocky Mounain spotted fever is often weakly positive, and proteus OX 2 and OXK are also sometimes agglutinated in low titre. On the basis of experience in many parts of the world, Zinsser and Bayne-Jones[6] believe that in true typhus fever the patient's serum develops an agglutination titre of from 1:100 to 1:200 by the end of the first week and that there is a gradual rise of the titre throughout the febrile period. In the most pronounced cases the titre may rise to 1:5000 although such high titres are relatively rare. Zinsser also believes that the titre in Rocky Mountain spotted fever does not go as high as it usually does in typhus. Gay et al[7] have found that the serum of typhus cases often agglutinates in titres of 1:2500 between the eighth and the twenty-first days of the illness.

Cross immunity tests using guinea pigs can be made to conclude the differentiation between typhus and spotted fever since immunization of a guinea pig against one of the diseases does not protect against the other. In the described case this method was not used as the characteristic rash and the diagnostic high titre of the Weil-

Felix reaction appeared to be conclusive. By communicating with the Florida State Health Department, we found that they had encountered no cases of Rocky Mountain spotted fever during the past two years, whereas typhus fever had been occurring fairly frequently, ten cases having been reported in December 1937 and January 1938 which coincided with the patient's visit to Florida.

CONCLUSIONS

A case of endemic typhus fever is presented as an unusual cause of unexplained fever which could not be diagnosed by the ordinary laboratory procedures. Only later in the course of the disease when the rash appeared was the diagnosis of endemic typhus fever made possible.

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REFERENCES

1. Maxcy, Kenneth F.: An Epidemiological Study of Endemic Typhus (Brill's Disease) in the Southeastern United States with Special Reference to its Mode of Transmission, Pub. Health Rep. U. S. P. H. Ser. **41**: 52, 2967, 1926.
2. Day, Alex A.: Personal communication.
3. McShane, John J.: State Epidemiologist, Illinois State Health Dept., Personal communication.
4. Rumreich, Adolf S.: The Typhus and Rocky Mountain Spotted Fever Group, J. A. M. A. **10**: 331-334, 1933.
5. Topley and Wilson: Principles of Bacteriology and Immunology, 1454-1470.
6. Zinsser and Bayne-Jones: Textbook of Bacteriology, 744-781.
7. Gay et al: Agents of Disease and Host Resistance, 1157.
8. Dyer, R. E.: Canad. Pub. Health Jour. **28**: 1-9, Jan. 1937.

THE PRACTICAL SIGNIFICANCE OF GROSS RECTAL BLEEDING

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Rectal bleeding is one of the more common complaints which general practitioners treat, yet it is a well-known fact that many of them take very little interest in the problem. Why is this so? Why are these physicians sufficiently interested in such symptoms as precordial pain, epigastric distress, sterility, hemoptysis and what not, to carefully study them, but not in rectal bleeding which therefore is neglected? Surely the symptoms are of equal importance to the patients and the patient with rectal bleeding is entitled to as much consideration and study as are the others.

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When a patient tells you he has vomited blood you do not look at his face or place a finger in his mouth and blindly try to make a diagnosis. That is stupid and very unscientific, but precisely the method used by many physicians in trying to find the cause of rectal bleeding. The patient assumes a knee-elbow position, the physician looks at the anus, perhaps finds an innocent skin tag, places a finger in the rectum and tries to feel an internal hemorrhoid which can't be felt. He isn't certain as to what he palpates and for want of a better diagnosis tells the patient he has "piles," prescribes mineral oil and suppositories and believes he has helped the patient. This negligent type of practice and the blind prescribing of local medicaments may lead to serious results and are to be condemned.

Just as the diagnosis of hematemesis entails obtaining a careful history, carefully examining the patient, having the gastric contents and the stools examined, taking gastrointestinal x-rays, and having gastroscopy performed; so to find the cause of rectal bleeding a physician must take a history of the complaints, thoroughly examine the patient and make a complete proctologic study. The latter includes proctosigmoidoscopy, examination of the stool and any rectal discharge, and x-raying the colon. The diagnosis cannot be properly made by any other method.

In the majority of cases gross blood that is passed per anum originates in pathological changes of the perianal region or lower alimentary canal. Occasionally its source is in the upper gastrointestinal tract. Urinary and genital hemorrhage cause confusion at times, but are easily differentiated by tamponing the urethral and vaginal orifices.

Rectal bleeding is a sign of a large number of conditions. Usually it signifies a benign lesion, but it also evidences a malignant one. Often the two coexist just a few inches apart. If this fact isn't kept in mind the malignancy beyond the reach of the finger will be missed unless a sigmoidoscopy is done. Only too frequently patients undergo hemorrhoidectomy while a carcinoma of the rectum or rectosigmoid which has been overlooked is permitted to progress from an operable to an inoperable stage.

To avoid this grave error, it is incumbent upon the physician to see that every patient with rectal bleeding receives a complete proctologic examination, which is not considered complete until

two questions are answered. These questions are: (1) what is the source of the bleeding, and (2) is a malignancy present.

There are three essentials to the proper diagnosis of gross rectal bleeding. These are (1) A knowledge of the anatomy of the anal canal and rectum; (2) A careful history, and (3) A general physical examination and a complete proctologic study. Briefly, the important anatomical markings are these: The perianal skin converges at the anus and continues as the lining of the anal canal. At Hilton's White Line the epithelium becomes transitional in type. Hilton's White Line is a linear depression which can be felt between the subcutaneous portion of the external sphincter and the internal sphincter, about half an inch proximal to the anus. Through the anoscope this depression appears whitish in color due to the connective tissue separating the sphincters and shining through the lining of the anal canal.

The juncture of the anal canal with the rectum is marked by the pectinate line and insertion of the levator ani muscle. The area between Hilton's White Line and the pectinate line is 6—9 mm. wide and was named the "Pecten" by Stroud[1] in 1896 from the resemblance to a comb. This is the site of the condition known as "Pectenosis."

The subcutaneous portion of the external sphincter muscle encases the distal portion of the anal canal, being on a plane with the internal sphincter which encircles the proximal portion of the canal. The latter is a continuation of the circular muscular coat of the rectum and is encased by the deep portion of the external sphincter. The longitudinal or external muscular coat of the rectum ends opposite the pectinate line in a fibromuscular septum which runs between the sphincters to be inserted into the lining of the anal canal.

Due to the purse-string action of the sphincters the rectal mucosa is thrown into vertical folds called columns of Morgagni. Their apices are at the pectinate line where each has a tiny mucosal projection called the "anal papilla." Between each column of Morgagni a small semi-lunar fold of mucosa is present. This is an anal valve, behind which is a small depression known as an anal crypt. Hellwig[2] has shown that a small duct opens into the majority of these crypts.

The rectum is divided by three valves—the rectal valves of Houston. The superior one is on the left, the middle one on the right and the inferior one on the left. These are capable of contracting and narrowing the rectal lumen. Similarly, a sphincteric mechanism exists at the rectosigmoidal juncture. This is known as O'Beirne's sphincter.

The pectinate line is an important landmark. Above it the blood supply is furnished by the superior hemorrhoidal artery and the middle hemorrhoidal arteries. Below it by the inferior hemorrhoidal arteries. Internal hemorrhoids originate above the pectinate line and external hemorrhoids below it.

The innervation is sympathetic above and cerebrospinal (S2, 3, 4, 5) below the line. Therefore, the ordinary pain sense is absent above the pectinate line and pain is less prominent clinically in lesions located there.

The second essential in the diagnosis of rectal bleeding is the history which is important because the details of the bleeding and the associated symptoms often point to the location of the lesion or the causative lesion itself.

What are the characteristics of gross rectal hemorrhage? The bleeding is either external or internal, mild or severe, and independent or associated with defecation, straining, or instrumentation. The blood is bright or dark red, mixed with the feces, found on the stool, paper, instrument, or underclothing, or in the water. Generally, the brighter red the blood is the lower its origin, and the darker, the higher its source. However, blood from a severely bleeding high lesion as a peptic ulcer, might be red when passed, while retained blood originating in the sigmoid can turn dark before being eliminated.

The associated symptoms include: pain, burning, itching, swelling, protrusion, prolapse, discharge, urgency and frequency of defecation, change in bowel habit, constipation, diarrhea, loss of weight, fever, anemia and leucocytosis.

The third essential—the proctologic examination—is the most important procedure in the diagnosis of rectal bleeding because it permits visualization and differentiation of the lesions.

It is much easier to obtain a history when one has some classification on which to base the questions. The anatomic position of the lesion is a practical means of classifying the various pathologic entities which cause rectal bleeding. This divides the lesions into five groups each of which

has in a general way its own type of bleeding and associated symptoms.

These groups are (1) the Perianal, (2) the Anal Canal, (3) the Pectinate, (4) the Rectal, and (5) the Colon.

Bleeding in the perianal group of lesions (Chart I) is external, bright red, and usually small in amount. Generally it is independent of bowel movement, developing in the course of some pathologic process or trauma. The blood is found on the paper, undergarment, or instrument, and occasionally on the stool or in the water. Pain and itching are concomitant symptoms.

CHART I

ANATOMIC CLASSIFICATION OF GROSS RECTAL BLEEDING

Group 1. Perianal Lesions.
Bleeding—Independent of Defecation; External.
Blood—Bright red; Small amount found on finger, Tissue, Garment.
Pain—Independent of Defecation.
Lesions 1. Thrombosed External Hemorrhoid ulcerated through skin tag.
2. Anal verge varicosity.
3. Pruritus ani.
4. Perianal abscess.
5. Perianal fistula.
6. Perianal wart.
7. Perianal ulceration.
8. Perianal wound.
9. Perianal surgical procedure.

The commonest perianal lesion is the external hemorrhoid, which does not bleed unless some complication develops. The most frequent complication is thrombosis, which may be followed by ulceration of the raised overlying skin tag. Then, with spontaneous extrusion of the clot, hemorrhage usually slight, but occasionally more severe, may result. Pain developing with the onset of the thrombosis disappears with extrusion of the clot. The diagnosis is made by inspection which reveals the external hemorrhoids and the ulcerated, bleeding skin tag beneath which a portion of the thrombus may be still present.

An anal verge varicosity bleeds only when an overlying fissure or ulcer extends or is torn into it, as can happen during straining or instrumentation. The bleeding may be profuse, defecation is painful, and examination discloses a tightly contracted anal sphincter, and the fissure at the base of which is the bleeding varicosity.

In pruritus ani bleeding is insignificant and the result of scratching the pruritic skin. The blood is found on the finger, paper or undergarment, and the diagnosis is made by observing the bleeding points in the fissured, denuded, excoriated, thickened, often weeping perianal skin.

External bleeding from a perianal abscess or an anorectal fistula is unimportant and appears when the abscess ruptures through the skin or is traumatized by instrumentation. Constant, increasing pain and swelling in the perianal region are the outstanding symptoms and are relieved by the discharge of the pus, which, mixed with a small amount of blood is seen coming from the external opening.

A perianal wart or ulceration bleeds when traumatized, and the hemorrhage from a perianal wound or surgical procedure does not present any diagnostic difficulty.

The next group of lesions are in the anal canal (Chart II) and differ from the perianal entities mainly in one respect—they bleed during bowel movement. Little blood is lost because the anal canal is closed immediately after defecation. Pain is prominent and more closely associated with bowel movement than in the perianal group.

CHART II

ANATOMIC CLASSIFICATION OF GROSS RECTAL BLEEDING

Group 11. Anal Canal Lesions.
Bleeding—Associated with defecation; external.
Blood—Bright red; Small amount, Found on feces, Tissue.
Pain—Prominent. Associated with defecation.
Lesions 1. Tear of normal lining of anal canal.
2. Tear of friable lining of anal canal.
3. Prolapsed anal mucosa.
4. Fissure in ano.
5. Stricture.
6. Epithelioma.

The forceful passage of a large, hard, rough stool may tear normal mucosa lining the anal canal to give pain and slight bleeding. The tear is easily detected through the anoscope. Similarly, foreign bodies and instrumentation may cause bleeding, which bowel movement aggravates by reopening the wounds.

As a result of infection or a chronic discharge the lining of the anal canal becomes friable and is easily traumatized during defecation or instrumentation. The bleeding that results is slight and the examination reveals the tear in the friable bleeding mucosa. In some cases varicosities are present and when these are involved by the trauma the hemorrhage is larger. The diagnosis is made by the presence of the bluish varicosities bleeding through the torn, friable mucosa of the anal canal.

The mucosa of the anal canal may become stretched by a subcutaneous hematoma or throm-

bus of an underlying varicosity. This stretched, redundant mucosa may prolapse through the anal canal, become friable and denuded, and will bleed if traumatized by stool or friction. The bleeding, eroded, or ulcerated, prolapsed mucosa is easily found on inspection.

The outstanding symptoms of an anal fissure is sharp pain during bowel movement, while bleeding usually is slight. The blood often streaks the stool. A sphincter spasm arouses suspicion of its presence and finding the ulcer with its sentinel pile makes the diagnosis.

About two per cent.[3] of the malignant growths in the lower 24 cm. of the bowel are in the anal canal. While excruciating pain is the most prominent symptom of an anal canal epithelioma, slight bleeding does occur with defecation and is the result of necrosis and trauma. The lesion may closely resemble a benign fissure but is differentiated by being somewhat fixed and indurated. Biopsy verifies the diagnosis.

The third group of lesions are in the pectinate zone (Chart III), duplicate the hemorrhage of the anal canal entities, and in addition bleed internally. Pain, burning, and itching accompany this group.

CHART III

ANATOMIC CLASSIFICATION OF GROSS RECTAL BLEEDING

Group III. Pectinate Lesions.
Duplicates Anal Canal Group in
Bleeding—Associated with Defecation.
Blood—Bright Red; Small Amount Found on Feces, Tissue.
Differs from Anal Canal Group in
Bleeding—Internally and Externally.
Pain—Less prominent.
Burning and itching with B. M.
Lesions 1. Cryptitis.
2. Papillitis.
3. Infected internal blind sinus.
4. Internal rupture of perirectal abscess.
5. Tuft of granulation tissue.
6. Stricture.
7. Torn anal valve.

Bleeding in cryptitis accompanies defecation, is slight and the result of trauma to be congested or eroded crypts. Burning and itching are frequently present, and the tender crypts appear injected, eroded, or ulcerated when viewed through the proctoscope.

Papillitis is often associated with cryptitis and produces identical symptoms. Trauma during defecation causes the swollen, injected and congested papillae to bleed. On palpation they are indurated, tender and enlarged, and through the proctoscope may be seen plastered down over adjacent inflamed crypts. Hypertrophic papillae may prolapse through the anal canal.

The mucosa surrounding the opening of an infected internal blind sinus is usually injected, and may bleed slightly when traumatized during defecation or instrumentation. The sinus is tender, and through the proctoscope one sees the injected, eroded mucosa around its opening from which pus may escape and into which a hook falls very easily. Pain, burning, soreness and itching are associated symptoms.

Bleeding occurs when a perirectal or perianal abscess ruptures internally, and usually is masked by a purulent discharge. The constant severe pain is alleviated, and the internal opening is found in a crypt.

In chronic, deep, inadequately draining abscesses and in fistulae a tuft of granulation tissue may form in the internal opening. This is very friable and bleeds very easily.

An obstructing, postoperative stricture may involve the anal canal, pectinate zone, or lower rectum, and will bleed if torn by instrumentation or defecation. Bleeding is external from an anal canal stricture and internal from the rectal and pectinate strictures. However, in the latter, bleeding can be external when the sphincters are incompetent. Frequently, bleeding is severe and immediate measures must be taken to prevent serious loss of blood.

Bleeding from a torn anal valve usually is mild but may be severe, collect in the rectum, and be passed as a clot with or without bowel movement.

The largest group of lesions causing hemorrhage are in the rectum (Chart IV) and present all of the characteristics of bleeding. Pain is less prominent in this group than in the pectinate, anal canal, and perianal types.

CHART IV

ANATOMIC CLASSIFICATION OF GROSS RECTAL BLEEDING

Group IV. Rectal Lesions.
This is the largest group. Bleeding is associated with defecation and presents all the characteristics of rectal hemorrhage.
Pain—Not prominent.
Tenesmus—Frequent.

Lesions:

1. Internal Hemorrhoid	11. Worms
2. Prolapsed Int. Hemorrhoid	12. Carcinoma
3. Proctitis	13. Adenoma
4. Prolapsed Rectal Mucosa	14. Lipoma
5. Procidentia Recti	15. Papilloma
6. Active Inflam. Stricture	16. Angioma
7. Healed Fibrous Stricture	17. Polypoidosis
8. Rectal Ulceration	18. Hypert. T. B.
9. Rectal Injury	19. Adenomyoma
10. Foreign Body	20. Prostate Ca.

The internal hemorrhoid is the commonest pathologic entity of the rectum. It is the most frequent cause of rectal hemorrhage, bleeding in 90 per cent. of the cases. At first, the patient discovers a small amount of bright red blood on the stool or paper, which through the proctoscope is seen to come from the eroded, ulcerated or traumatized, friable mucosa covering the hemorrhoidal varicosity. With ulceration into the venous spaces, the bleeding becomes more profuse, at times continuing after defecation to be passed later as a blood clot. Diarrhea, constipation, and straining aggravate the bleeding.

Prolapse of the internal hemorrhoid leads to further vascular disturbance, more erosion and ulceration, and more bleeding, which usually ceases soon after it is returned into the rectum.

The bleeding in proctitis usually is slight unless associated with a colitis, and through the proctoscope is seen to be due to mucosal erosion or injury to the mucosal vessels. The patient complains of heaviness in the rectum with tenesmus, urgency and frequency of defecation.

In prolapse of the rectal mucosa and in procidentia recti bleeding is usually slight coming from erosions, ulcerations, or traumatic lesions of the mucosa, and ceases soon after reduction.

The bleeding associated with an active, inflammatory rectal stricture is part of mucopurulent discharge and somewhat dark in color. Healed fibrous rectal strictures bleed only when traumatized in which case bright red blood is passed.

Bleeding from rectal ulceration is dependent upon the number of vessels involved and the amount of damage to them. The blood may be passed with stool or as part of a purulent discharge which may be present. Through the sigmoidoscope one may see that the ulcerative process in the rectum is part of a similar one involving the colon.

The hemorrhage from rectal injuries varies with the type and extent of the wounds. Blood that collects in the rectum is eliminated immediately or after some delay. If the peritoneum is opened bleeding occurs into the peritoneal cavity. Similarly, hemorrhage may take place into the bladder, vagina and other organs. Many wounds are easily seen through the proctoscope, but some can only be found by laparatomy.

The bleeding from foreign bodies in the rectum is traumatic, and often more difficulty is encountered in treatment than in diagnosis.

When worms cause bleeding they do so by traumatizing the mucosa. Usually they are scattered throughout the colon, being especially numerous in the cecum. Therefore, bleeding which may be diffuse is more marked proximally where the blood can easily mix with the stool. The worms may be seen on the stool or through the sigmoidoscope on the eroded mucosa.

Carcinoma of the rectum is not the most frequent but the most important cause of rectal bleeding due to its seriousness. Bleeding is not only its commonest sign, being present in 90 per cent of the cases, but also its initial sign 33 per cent of the time.[4] A small amount of blood may be passed with the stool or an alarming hemorrhage may ensue. Irregularity of bowel habit, pain and blood in the stools should arouse suspicion of its presence, and the diagnosis is made by finding the fixed, indurated, somewhat tender, friable tumor mass that often is centrally necrotic and ulcerated, and bleeds upon the slightest trauma. Biopsy verifies the diagnosis. Eighty per cent of the carcinomas are within finger reach and an additional number can be seen through the sigmoidoscope. Thus the importance of a complete proctologic examination in rectal bleeding becomes clearly evident.

In the group of benign and rare tumors of the rectum are four with the tendency to bleed. The adenoma, which originates in a gland of Lieberkuhn is the commonest and the most important because it frequently undergoes malignan degeneration. It constitutes sixty-three[5] to eighty [6] per cent of the tumors of the rectum and may or may not cause symptoms. Bleeding with defecation is its most frequent sign resulting from ulceration, trauma and malignant degeneration. The loss of blood usually is slight, spotting the stool, but may be moderate with prolapse, and alarming with spontaneous amputation of the adenoma.[7] On proctoscopic examination one finds the sessile or pedunculated, grapelike, pink to dark red, ulcerated or bleeding tumor.

The second most common benign tumor is the lipoma which arises from the subserosa or submucosa. It, too, may be symptomless, but bleeding is a frequent and often initial sign incident to ulceration of the overlying mucosa or tumor itself. Through the proctoscope one may see the ulcerated, elevated mucosa, or the sessile or

pedunculated, lobulated, rounded, or ovoid tumor that is ulcerated when bleeding is present.

The third tumor, the papilloma, is rare and originates in the intestinal mucosa in a crypt of Lieberkuhn. Its outstanding symptom is bleeding, though large growths may be accompanied by tenesmus and the frequent discharge of mucus. The tumor is sessile, being composed of many closely set villi that show ulcerations.

The fourth tumor, the angioma, is one of the rarest tumors of the rectum. It apparently is congenital arising from the submucosa. Ulcerations may lead to fatal hemorrhage and the diagnosis is made by the proctoscopic finding of a purplish tumefaction often surrounded by small areas resembling nevi.

The multiple adenomas of polyposis of the rectum bleed when they are ulcerated, traumatized, or undergo malignant degeneration, and present little diagnostic difficulty. The patient complains of bleeding with defecation or often, frequent bloody mucoid discharges. The numerous grapelike growths of varied size, shape, and color seen through the proctoscope can't be confused with anything else.

When bleeding is present in hypertrophic tuberculosis of the rectum it usually is slight and due to ulceration of the covering mucosa. The lesion simulates a carcinoma and biopsy is necessary for differentiation.

An adenomyoma of the rectovaginal septum that has ulcerated through the rectal wall bleeds during the menstrual period. Constipation is a concomitant symptom. The ulcerated, bleeding mass in the rectovaginal septum is moderately fixed and differentiated from carcinoma by biopsy.

Rectal bleeding from a prostate carcinoma that has extended through the rectal mucosa is due to ulceration and trauma. Urinary symptoms usually precede the rectal bleeding, and the diagnosis is made by finding the extra rectal mass fixed to the prostate and ulcerated into the rectum. Biopsy substantiates the diagnosis.

There are two outstanding characteristics of bleeding associated with pathological findings in the colon (Chart V) namely: (1) the hemorrhage is internal, and (2) the blood is mixed with the stool. Diarrhea is frequently present and cramps due to spasms of the colon may be very severe.

CHART V

ANATOMIC CLASSIFICATION OF GROSS RECTAL BLEEDING

Group V. Colon Lesions.
Bleeding is internal.
Blood is bright or dark red, mixed with the feces and often passed as part of a mucopurulent discharge.
Diarrhea and cramps frequent.
Lesions 1. Acute Bacillary dysentery.
2. Amoebic dysentery.
3. Chronic Thrombo-Ulc. colitis.
4. Tuberculous colitis.
5. Diverticulitis.
6. Intussusception.
7. Carcinoma of left colon.
8. Carcinoma of right colon.
9. Metallic poisoning.
10. Non-specific granulomatous colitis.

Blood forms a conspicuous part of the mucopurulent discharge seen in acute bacillary dysentery. At the onset it is bright red because the colon is emptied at frequent intervals. Later, as the number of discharges lessen, it becomes darker, and with healing, gradually disappears. The feces are liquid and mixed with the discharge. Toxemia often is marked and diarrhea, cramps and tenesmus are prominent. The mucosa as seen through the sigmoidoscope is intensely injected and covered with innumerable, pinpoint to a few millimeters in diameter ulcerations with hemorrhagic and purulent bases, and the slightest trauma produces severe bleeding. Culture and agglutination tests identify the causative organism.

In general, there is less blood in the discharges of amoebic dysentery than in bacillary dysentery. The toxemia, too, is usually milder. Viewed through the sigmoidoscope, the typical mucosal ulcers are buttonhole or umbilicated being circular or elliptical, 5 to 10 mm. in diameter with sides elevated up to 2 mm. and bases yellowish and necrotic. The mucosa between the ulcers is usually quite normal. The amoebae or cysts are found in the stool, discharge or smear from the base of the ulcer.

Chronic ulcerative colitis is characterized by a bloody mucopurulent discharge and diarrhea. Through the signmoidoscope one sees that the mucosal picture varies with the stage of the disease. Early it is granular, injected, and covered with innumerable pinpoint to pinhead sized hemorrhagic ulcerations in between which are minute mucosal abscesses. Later, larger, irregular ulcers and small steelate scars may be present.

In tuberculous colitis bleeding is mild and often occult. Diarrhea may be present and the patient has an advanced pulmonary tuberculosis.

Sigmoidoscopically, the ulcers are a few millimeters to several centimeters in diameter, irregular, and have slightly raised, reddened, and often undermined edges. Their bases are covered with yellow pyogenic membranes beneath which tuberculous granulation tissue is found. The mucosa may present vascular changes.

Rectal bleeding is present in a small number of the cases of diverticulitis. Usually it is mild and due to fecal traumatization of the congested mucosa in the region of the diverticula. The history of left lower quadrant pain, constipation and a mass in the region of the sigmoid point to the diagnosis which is confirmed by sigmoidoscopy and x-ray examination.

Bleeding is of diagnostic importance in intussusception, usually appearing within 12 hours following the onset of the severe cramps and shock which seize the child. The bleeding results from venous congestion and necrosis due to interference with circulation, and the blood is passed alone or with some mucus. The history and presence of a tumor suggest the diagnosis and the intussusception may be seen through the signmoidoscope.

In carcinoma of the left side of the colon, bleeding is prominent due to the firm character of the intestinal content. Often the blood is bright red and found on the stool. Symptoms of obstruction are early in appearance and the sigmoidoscopic or x-ray demonstration of the single, fixed, indurated, often ulcerated mass with a necrotic center makes the diagnosis which biopsy confirms.

In carcinoma of the right side of the colon bleeding is usually slight and occult, gross hemorrhage being rare and dependent upon ulceration. Dyspeptic symptoms, change in bowel habit, anemia and a mass in the right side of the abdomen suggest the condition which is confirmed by x-ray, exploratory, and biopsy.

In metallic poisoning the amount of bleeding depends upon the severity of the damage and extent of the erosions and ulcerations in the mucosa. Bright red and dark blood is passed with mucus and diarrheal stools. The severe nausea, vomiting, cramps, and prostration, point to the diagnosis which chemical tests prove.

Colitis of the right side of the large bowel is found in approximately five per cent of the cases of non-specific granulomas of the ileocecal region.[8] In this disease the loss of blood in the stool is insignificant and occult, the important symptoms being diarrhea, abdominal pain, fever and mass formation. X-ray examination and laparotomy make the diagnosis.

Bleeding is associated with the following conditions which are not in the scope of this discussion, but are included for completeness: carcinoma of the small bowel, typhoid fever, tuberculous enteritis, small bowel injuries, ectopic gastric mucosa, mesenteric thrombosis or embolism, peptic ulcers of the duodenum and stomach, carcinoma of the stomach, gastritis, rupture of an aneurysm into the stomach or esophagus, carcinoma of the esophagus, swallowed blood, pellagra, chronic heart disease with portal congestion, chronic interstitial nephritis with uremia, severe secondary anemia, leukemias, purpura hemorrhagica, pernicious anemia, and septic infection of the umbilical cord in infants.

Summarizing, in the majority of cases, gross blood that is passed per anum originates in lesions of the perianal region or lower alimentary canal. For practical purposes these pathologic entities are classified according to their anatomic positions. Therefore, the following groups are differentiated each having its own general type of bleeding and associated symptoms: the perianal, with external bleeding generally independent of bowel movement; the anal canal, with a small amount of external bleeding associated with defecation; the pectinate, with external bleeding associated with bowel movement and in addition internal bleeding; the rectal in which the bleeding presents all the characteristics of hemorrhage per anum and in which pain is less prominent than in the preceding groups; and last, the colon, in which the bleeding is internal, the blood is mixed with the stool, and diarrhea is frequently present.

Finally, one should remember that the investigation of rectal bleeding demands a complete proctologic examination to find the source of the hemorrhage and to determine the presence or absence of a malignancy.

REFERENCES

1. Stroud, Bert, B.: On the Anatomy of the Anus, Annals of Surgery. **24**: 1, 1896.
2. Tucker, C. C. and Hellwig, A.: Anal Ducts, Comparative and Developmental Histology, Arch. Surg. **31**: 521, 1935.
3. Buie, Louis A., and Brust, John C. M.: Malignant Anal Lesions of Epithelial Origin. Jour. Lancet **53**: 565, 1933.
4. Rankin, Bargen, and Buie: The Colon, Rectum, and Anus, Philadelphia, W. B. Saunders Co. P. 528, 1935.
5. Erdmann and Morris: Polyposis of the Colon, S. G. O. 460, Sept., 1928.
6. Dewis, J. W.: A small fibroma of the ileum resulting in obstruction of the bowel, with a consideration of various forms of benign intestinal tumors. Boston Med. and Surg. Jour. **155**: 427-433, 1906.

7. Diamond, Mortimer: Adenoma of the Rectum in Children, Am. Jour. Dis. Child. 56: 360, 1939.

8. Crohn, B. B.: Non-specific granulomatous lesions in and about the ileocecal region, J. Med. 19: 84-87, 1938.

58 East Washington Street.

HOT DOGS, COLLEGE AND DEPRESSION

Once there was a man who lived by the side of the road and sold hot dogs.

He was hard of hearing, so he had no radio. He had trouble with his eyes, so he read no newspapers. But he sold good hot dogs. He put signs up on the highway, telling how good they were. He stood on the side of the road and cried, "Buy a hot dog, Mister?" And the people bought.

He increased his meat and bun orders. He bought a bigger stove to take care of his trade. He finally had his son come home from college to help him.

But then something happened. His son said, "Father, haven't you been listening to the radio? Haven't you been reading the newspapers? There's a big depression on. The European situation is terrible. The domestic situation is worse. Everything's going to pot."

Whereupon the father thought, "Well, my son's been to college, he reads the papers, and he listens to the radio, and he ought to know." So the father put up no more advertising signs, cut down on his meat and bun orders, and no longer bothered to stand out on the highway to call his wares. And his hot dog sales fell off almost overnight.

"You're right son," the father said to the boy. *"We certainly are in a Great Depression."*—Paul Talbot. Courtesy of United Business Service.

EXAMINATIONS

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next written examination and review of case histories (Part I) for Group B candidates will be held in various cities of the United States and Canada on Saturday, January 6, 1940, at 2:00 P. M. The Board announces that it will hold only one Group B, Part I, examination this year prior to the final general examination (Part II), instead of two as in former years. Candidates who successfully complete the Part I examination proceed automatically to the Part II examination held in June, 1940.

Applications for admission to Group B, Part I, examinations must be on file in the Secretary's office not later than October 4, 1939.

The general oral and pathological examinations (Part II) for all candidates (Groups A and B) will be conducted by the entire Board, meeting in Atlantic City, N. J., on June 8, 9, 10, and 11, 1940, immediately prior to the annual meeting of the American Medical Association in New York City.

Applications for admission to Group A, Part II examinations must be on file in the Secretary's office not later than March 15, 1940.

DURATION OF SMALLPOX IMMUNITY

In 1936 a study was made of smallpox immunity in 1,053 matriculating college students at Kansas State College. In September 1937 David T. Loy and M. W. Husband, Manhattan, Kan. (*Journal A. M. A.*, Aug. 27, 1938), made a similar investigation of 986 students with additional data on the duration of smallpox immunity and the effect of multiple vaccinations. In summary they state: 1. Of the students who matriculate at Kansas State College (*a*) approximately one-fourth have never been vaccinated against smallpox and (*b*) approximately two-thirds are in some degree susceptible to smallpox. 2. Only 5.9 per cent. of the 1937 group had been vaccinated more than once previously. 3. There are about 20 per cent more persons with complete immunity in the previously vaccinated one to five year group than in the group vaccinated from six to ten years previously. 4. There are about 20 per cent. more persons with complete immunity in the multiple previously vaccinated group than in the once previously vaccinated group. 5. The method of noncompulsory vaccination used has proved to be almost 100 per cent. acceptable to the student group.

MALE HORMONE AND DESCENT OF TESTES

Hamilton, J. B., Anatomical Record, April, 1938. Has seen cryptorchid testes of the immature macaque descend into the scrotum following adequate administration of testosterone acetate and testosterone propionate. There was less edema and scrotal swelling than in descent produced by gonadotropic substance. Processes considered largely responsible in the production of this descent are (1) growth and elongation of the elements of the cord and (2) slight development of the scrotum to form more of a pouch for the testes. Descent produced by anterior pituitary-like substances may be due in part to stimulation of the secretion of male hormone. Endocrine treatment may be of value in (1) producing descent in certain cryptorchid cases, (2) authorizing surgery at an early age instead of waiting until puberty to see whether spontaneous descent may occur and (3) aiding surgery preoperatively by the development of structures of the cord and postoperatively by preventing retraction and tension.

BUSINESS GIRL

The amiable old gent visiting a home was trying to win the favor of the small daughter. "I'll give you a nickel for a kiss," he said. "No, thank you," she replied sweetly. "I can make more money taking castor oil."—*Brotherhood Progress.*

Marriages

ABE R. EVELOFF, Springfield, Ill., to Miss Vivian Grebler at St. Louis, July 13.

THEODORE SIDNEY RAIFORD to Miss Ann Irish, both of Decatur, Ill., June 20.

Personals

Dr. James J. Callahan, Chicago, discussed fractures before the Effingham County Medical Society in Effingham, June 13.

Arnold F. Emch, Ph.D., has resigned as executive director of the Chicago Hospital Council to become assistant secretary of the American Hospital Association.

Dr. Clement C. Clay, medical assistant to the director, University of Chicago Clinics, has resigned to become administrator of St. Barnabas Hospital, Minneapolis, succeeding Samuel W. Rice, resigned.

At a joint meeting of the Fulton and McDonough county medical societies in Bushnell June 20 Drs. James C. Redington and Frederic E. Hambrecht, Galesburg, spoke on "The Diagnosis of Pulmonary Tuberculosis" and "Surgical Treatment of Pulmonary Tuberculosis" respectively.

Sr. Surg. William H. Slaughter has been appointed medical officer in charge of the U. S. Marine Hospital, succeeding Dr. Mark J. White, who retired May 1. Dr. Slaughter comes from a similar post in New Orleans, where he was succeeded by Dr. Lionel E. Hooper, Honolulu.

At a meeting of the Madison County Medical Society in Alton August 4 the speakers were Drs. Isaac A. Abt, Chicago, on "Infant Feeding First Two Years"; John F. Carey, Joliet, "Treatment of the Sick Infant," and Franklin J. Corper, Chicago, "Respiratory Tract Infections in Children."

Dr. Herbert A. Potts, for many years professor of oral surgery at Northwestern University medical and dental schools, will retire from the dental school at the end of the academic year September 1, with the title of professor emeritus. Dr. Potts, who also has the degree of doctor of dental surgery, has been associated with Northwestern University Dental School since 1908, first as lecturer in anesthesia, assistant in oral surgery, professor of pathology, instructor in oral surgery, assistant professor and since 1921 professor of oral surgery. He received his degree in dentistry at Northwestern in 1895 and his medical degree in 1901. He retired from the medical school as professor emeritus June 30, 1938.

Dr. Morgan J. O'Connell has been invited to address the Rock Island County Maternal Welfare Committee August 22, subject, "The Indications, Contraindications and Technique of Forceps Delivery."

Dr. C. C. Maher addressed before the Henry County Medical Society, August 24, on the subject of "Cardio Renal Disease."

Dr. Guy Cushing was invited to speak before the Henry County Medical Society, August 24, on the subject of "Acute, Perforating Gastric and Duodenal Ulcers."

Dr. Donald A. R. Morrison, formerly Rockefeller Fellow at the Pennsylvania Hospital for Mental and Nervous Diseases, and for the past two years instructor in Psychiatry at the University of Chicago Medical School, has joined the staff of the Rogers Memorial Sanitarium, Oconomowoc, Wis.

During the year ending April, 1939, 209 members of the Chicago Medical Society presented scientific papers before downstate county medical societies at the request of the Scientific Service Committee of the Illinois State Medical Society. Many others appeared who were not scheduled through the main office.

Franklin J. Corper and Isaac Abt presented a program on "Infections of the Respiratory Tract in Chidren" and "Infant Feeding" before the Madison County Medical Society, August 4.

News Notes

—There were 2,748 deliveries without a maternal death in the Chicago Lying-In Hospital of the University of Chicago during the fiscal year ended June 30. In 571 deliveries of the hospital's home service there was but one death.

—To determine the effectiveness of efforts toward the control of venereal diseases, a resurvey of all treatment sources is being planned in accordance with a request from Dr. Thomas Parran, surgeon general, U. S. Public Health Service, according to the Chicago Medical Society *Bulletin*. A campaign was begun throughout the state in June, 1937, to bring patients with syphilis under medical care.

—A survey course for persons actively engaged in the administration of hospitals will be offered evenings at University College, University of Chicago, during the autumn and winter, in cooperation with the American College of

Hospital Administrators. Among the lecturers will be: Drs. Robin C. Buerki, president of the college of hospital administrators; Bert W. Caldwell, executive secretary, American Hospital Asciation; Malcolm T. MacEachern, associate director, American College of Surgeons, and Herman Smith, medical superintendent of Michael Reese Hospital.

—The Institute of Medicine of Chicago offers again the Joseph A. Capps Prize of $400 for the most meritorious investigation in medicine or in the specialties of medicine. Competition is open to graduates of medical schools in Chicago who have completed an internship or a year of laboratory work in 1937 or thereafter. The investigation may also be in the fundamental sciences provided the work has a definite bearing on a medical problem. Manuscripts must be submitted to the secretary of the institute, 86 East Randolph Street, Chicago, not later than December 31.

—State fair visitors were invited to have free blood tests made by the state department of health during the week of the fair, August 12-19. Code numbers were used instead of names in order to keep the results confidential. A special laboratory was established on the grounds and the visitors were invited to see the process and hear it explained by a lecturer. The department also conducted a "better babies conference" and presented numerous exhibits and demonstrations. Among the latter were an exhibit on pneumonia, models showing care of patients with contagious disease, a modern swimming pool and a giant tooth showing principles of dental hygiene.

—The cornerstone of the Neuropsychiatric Institute, University of Illinois College of Medicine, was laid with appropriate ceremonies recently. Addresses were delivered, among others by Arthur Cutts Willard, LL.D., president, University of Illinois; D. R. Kennicott, regional director, PWA; Dr. Richard H. Hutchings, Utica, N. Y., and Dr. Adolph Meyer, Baltimore. The cornerstone was laid by A. L. Bowen, director of the state department of public welfare, and the invocation given by Rev. Arthur E. Johnstone of the Episcopal City Missions. The building is expected to be ready for occupancy in the fall of 1940. More than $500,000 of the approximate cost of $1,300,000 has been supplied by PWA funds. The building, which will be eleven stories high, will be used for teaching neurology and psychiatry and for research in these fields.

—The state department of health has issued a study of mortality trends from 1925 to 1939. The general death rate for 1925 was 11.5 per thousand of population and for 1938 it was 10.7. Infant mortality has steadily declined: in 1927 it was 64 per thousand live births and in 1938 it was 41. Deaths of mothers declined from 5.5 per thousand births in 1925 to 3.1 in 1938. Heart disease, cancer and diabetes have increased, but there are more persons in the population of an age to suffer these diseases. The rates from heart disease in 1925 and in 1938 were 186.9 and 313.5 per hundred thousand, respectively. Most of the increase occurred among persons of the higher age levels. Deaths from cancer rose, a rate of 102.2 per hundred thousand in 1925 to a rate of 136.4 in 1938. Diabetes increased from 19.7 to 27.6 per hundred thousand of population.

—A total of 505 patients representing twenty-two states, Canada, Cuba, South America and Spain were treated at the Chicago Tumor Institute during its first year of operation, according to its initial report recently released. Seventeen indigent patients were cared for without any referring physicians. There were 4,772 examinations made during the year: 1,299 x-ray diagnostic procedures and 1,975 treatments given with radium and 3,576 with x-ray. There were about twenty-five classifications of the types of cancer. Leading the list were: cancer of the breast, 104 cases; larynx, forty-three; cervix and uterus, twenty-six each, and tongue, twenty-three. The report acknowledges the arrangement with the veterans' hospital at Hines which has enabled the institute to utilize the clinical material at Hines to project special research on cancer of the larynx. The report emphasizes the need of an experimental laboratory to supplement the clinical investigations. Space is available but funds do not permit this activity. Another need of the institute is a library of current periodicals and monographs on cancer. During the year postgraduate instruction was given to about fifty specialists from all parts of the United States and from Hawaii, Cuba and South America. The teaching program of the institute is being developed on a comprehensive scale. The report classifies the program

as follows: one year fellowships or traineeships; visiting fellows (from one to six months); postgraduate instruction to radiologists (from one to three months); short annual courses (one week) and weekly cancer clinics for fourth year students at Northwestern University Medical School (ninety students). The Chicago Tumor Institute, opened in March, 1938, was chartered in Illinois, not for profit, to conduct research on the causes, diagnosis and treatment of cancer and to train cancer specialists. The institute has available 11 Gm. of radium, 10 Gm. of which is used in the form of a radium bomb. Dr. Max Cutler is director of the institute and Dr. Ludwig Hektoen is president of the board of trustees.

The McLean County Medical Society will hold a meeting at the Illinois Hotel, Bloomington, Illinois, on September 21. Dr. Rock Sleyster, President of the American Medical Association, will speak on "Medical Problems of the Day" and Dr. M. Herbert Barker of Northwestern University Medical School will speak on the subject of "Hypertension." Physicians of central Illinois are invited to attend this meeting and reservations for the dinner should be made with Dr. H. P. Sloan of Bloomington who is in charge of the arrangements.

Through a grant of $3,000 from the Julius Rosenwald Fund to the University of Chicago, x-ray examinations of approximately 30,000 patients of the Provident Hospital Clinic will be made during the next year to ascertain the extent of unsuspected tuberculosis. Announcement of the grant was made yesterday by Frederic Woodward, Vice President of the University.

Investigations recently made at the University of Chicago indicate that tuberculosis of the lungs is being overlooked to a surprising degree. At least one and one-half per cent. of the supposedly healthy population of the United States have clinically significant tuberculosis of the lungs without any awareness of their condition, the University study indicates. General complacency over the conquest of tuberculosis, the investigations suggest, may not be entirely justified.

The flouroscopic examination of a large group of patients through Provident Hospital Clinic will give sufficient statistical information for determination of the extent of unsuspected tuberculosis. All patients registered will be examined, and the families of all those found to have the disease also will be examined. The Rosenwald Fund gift will provide equipment for the examinations.

Deaths

HARRY HOMER DALLY, Chicago; Rush Medical College, Chicago, 1902; aged 60; died, May 20, in Kempton of cerebral hemorrhage.

HUGH JOHN DUFFY, Chicago; University of Illinois College of Medicine, Chicago, 1911; served during the World War; aged 50; on the staff of St. Francis Hospital, Evanston, Ill., where he died, May 8.

CHAUNCEY E. EHLE, Quincy, Ill.; Northwestern University Medical School, Chicago, 1895; member of the Illinois State Medical Society; superintendent of the Illinois Soldiers' and Sailors' Home and Hospital; died, May 23, in the Blessing Hospital.

THOMAS C. HAINLINE, Seaton, Ill.; Keokuk (Iowa) Medical College, 1897; age 81; died, May 19, of an injury received in a fall.

ARTHUR N. HOUSE, Kankakee, Ill.; Rush Medical College, Chicago, 1893; aged 72; died, May 12, near Escalon, Calif., of arteriosclerosis and heart disease.

MYRON ELLIS KAHN, Chicago; University of Illinois College of Medicine, Chicago, 1915; member of the American Academy of Ophthalmology and Oto-Laryngology; fellow of the American College of Surgeons; served during the World War; on the staffs of the Michael Reese and Sarah Morris hospitals and the Winfield (Ill.) Sanatorium; aged 47; died, May 25.

GEORGE T. LOVE, Wenona, Ill.; Hospital College of Medicine, Louisville, Ky., 1897; a Fellow, A. M. A.; aged 66; died, May 1, in St. Mary's Hospital, Streator.

JOSEPH F. MILLER, Palmer, Ill.; Missouri Medical College, St. Louis, 1889; a Fellow, A. M. A.; for many years bank president; aged 82; died, May 6, in St. Vincent Hospital, Taylorville, of pneumonia.

LINNAEUS HODGSON PRINCE, Hines, Ill.; Jefferson Medical College of Philadelphia, 1900; member of the American Association of Pathologists and Bacteriologists; served during the World War; served as pathologist on the staff of the Veterans Administration Facility, where he died, July 8, of arteriosclerosis and cerebral hemorrhage.

CARL J. F. ROCHOW, Rock Island, Ill.; Rush Medical College, Chicago, 1903; a Fellow, A. M. A.; aged 61; on the staff of St. Anthony's Hospital, where he died, May 11, of heart disease.

ALBERT RUBLY TRAPP, Lincoln, Ill.; Rush Medical College, Chicago, 1901; a Fellow, A. M. A., served during the World War; aged 62; died, May 8, in St. John's Hospital, Springfield, of infection of the prostate and bladder.

ARTHUR EDWARD WILLIAMS, Rock Island, Ill.; Northwestern University Medical School, Chicago, 1902; member of the Illinois State Medical Society; past president of the Rock Island County Medical Society; aged 63; on the staff of St. Anthony's Hospital, where he died, May 25, of heart disease.

GIFFORD DEAN WRAY JR., Chicago; School of Medicine of the Division of Biological Sciences of the University of Chicago, 1939; aged 24; was found dead, May 2, of poison, self administered.